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DERMATOLOGIC-DENTAL RELATIONSHIPS*

M. H. SAMITZ, M.D., JACK WEINER, M.D., and ALFRED B. MILLER, M.D.†

Specialization in medicine and in dentistry has had effective results in advancing our knowledge and for improving the health of mankind. Unfortunately, one of the well recognized drawbacks of most specialties has been the rather strict manner in which they have focused their attentions on only the very narrow aspects of their individual specialties. The responsibility of the dermatologist is not fulfilled if he devotes himself exclusively to the skin itself but his perspective must be expanded wherein the skin is correlated with other organic or functional disturbances and wherein he can interpolate the clinical and laboratory experience of other organ systems with the skin. Our endeavor should be focused upon overcoming the deficiency of specialization by developing an integrated approach which embraces the complete health picture of the patient. This challenge can be met if specialists in each narrowly prescribed field of research or clinical practice can integrate their separate fragments of knowledge for the mutual benefit of science and mankind.

The importance of this program is well recognized and it is noteworthy to mention the role that dentistry plays in this cooperative effort. The dentist associated with a good dermatologic service does not run a dental clinic—the dental surgeon should be an integral unit of the tumor team which includes the radiologist, plastic surgeon, pathologist, and dermatologist; and the dental physician should be part of a panel of the correlated medical-dermatologic conference which includes the internist, hematologist, pathologist, endocrinologist, psychiatrist, and peripheral-vascular consultant. In addition, investigative problems frequently will require the cooperation of the dental-medical staff. An example of coordination of investigational and clinical problems can be demonstrated in the dermatologic-allergy clinic where the dentist is given the opportunity to construct appliances suitable for patch testing in the oral cavity. The point we are trying to stress is that the limitation of dental consultation to treatment of associated oral disease as a separate and unrelated entity is shortsighted. The modern dentist, in order to discharge his obligation intelligently, should integrate his special knowledge of infection, immunology, physiology, and nutrition. Thus the new trend in medicine is inaugurated, namely a change in concept from curative to preventive medicine.

This review is an attempt to coordinate the specialties of dermatology and dentistry on a clinical level. It would be advisable if this study could be projected to include physiologic and physicochemical relationships, however this task would be too pretentious an undertaking for the short time allotted for this paper.

The following outline is an oversimplified description—accurate and adequate enough for our present purposes—of the causal mechanisms responsible for dermatologic diseases and their dental counterparts. Dental relationships will be discussed along two avenues: (1) oral manifestations of dermatoses—those skin conditions which show major oral manifestations, and (2) associated mouth and skin manifestations of systemic diseases. To complete the picture of dermatologic-dental relationships, the following subjects will also be reviewed: (1) occupational dermatoses of dentists, (2) occupational stigmata of the mouth and teeth, (3) cosmetics for the teeth and mouth, (4) fluorescence of the mouth, and (5) dryness of the mouth.

DERMATOLOGIC DISEASES

(I) Congenital

(II) Of External Origin

(III) Of Internal or Systemic Origin

(IV) Exclusive Dermatologic Entities of Known or Unknown Origin

* The material in this paper was used as the basis for a talk, which with 100 projected slides, was presented at the 8th Annual Meeting of the American Academy of Dental Medicine, May 14-16, 1954, Philadelphia, Pa.

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(I) Congenital	*Oral Manifestations*
Congenital ectodermal defects	Absence of teeth, deformities.
Congenital porphyria	Erythrodontia.
Epidermolysis bullosa	Bullae on mucous membranes of mouth and tongue.
Prenatal syphilis	Rhagades, Hutchinson incisors, mulberry molars.
Osler's Disease	Telangiectasia, angiomas.
Pachonychia congenita Bourneville's Syndrome (tuberous sclerosus, adenoma sebaceum)	Leukokeratosis of tongue, hypertrophy of interdental papillae; scars of tongue resulting from tongue biting in epileptic seizures.
Cheilitis glandularis apostematosa	Involvement of lower lip.

(II) Of External Origin	*Oral Manifestations*
Trauma	Traumatic ulcers, scars, angular cheilitis.
Fungus diseases superficial deep	Moniliasis: perleche, glossitis Lesions of actinomycosis, blastomycosis, histoplasmosis, torulosis, etc.
Virus diseases	Lesions of herpes simplex, Kaposi's varicelliform eruption, herpes zoster, foot and mouth disease, verrucae, molluscum contagiosum.
Parasitic and Protozoal diseases	Chaga's Disease (trypanosoma) Trichinosis: swelling of tongue, edema of soft palate, hemmorhages. Amebiasis cutis.
Bacterial diseases	Staphylococcic and streptococcic infections: pyodermas, pyogenic granulomas particularly following apical abscess, perleche, stomatitis, gingivitis.

(III) Of Internal or Systemic Origin	*Oral Manifestations*
Infectious diseases	
(a) Exanthemata: measles, scarlet fever, varicella	Koplik spots; strawberry tongue, etc.
(b) Other viral and bacterial diseases	Acute infectious gingivostomatitis, diphtheria, tularemia, glanders.
(c) Specific granulomas—tuberculosis, syphilis, leprosy, sarcoidosis	Tuberculosis cutis orificialis, chancre, gumma.
Drug eruptions	
(a) Drug sensitivity	Gingival hypertrophy (dilantin), fixed drug eruption, etc.
(b) Drug intolerance and irritancy—bismuth, lead, chromic acid, fluorine	Bismuth and lead lines, chrome ulcers, mottling of tooth enamel in fluorosis.
Urticaria and Erythema multiforme	Angioneurotic edema, Stevens-Johnson syndrome, etc.
Collagen diseases: scleroderma, lupus erythematosus periarteritis nodosa	Tongue, gum, and buccal mucosa lesions.
Metabolic diseases;	
(a) Amyloidosis	(a) Glossitis, macroglossia
(b) Lipoid proteinosis	(b) Wood-like tongue, bound to floor of mouth
(c) Xanthomatoses	(c) Yellowish papules on palate, etc.
(d) Hand-Schüller-Christian	(d) Gingivitis, stomatitis, etc.
(e) Extracellular cholesterosis	(e) Erythema multiforme-like papules on mucosa of mouth, soft palate.

	Oral Manifestations
(f) Diabetes, etc.	(f) Inflamed, turgid gums and sub-lingual tissues, enlarged, raw-ham colored tongue, dry and burning sensations, etc.
Nutritional and Vitamin deficiency diseases	
Vitamin B group—thiamine hydrochloride	Herpetic lesions and aphthae, sensitive and painful oral tissue.
riboflavin	Perleche, gingivitis, glossitis.
nicotinic acid	Smooth atrophy of lateral borders and tip of tongue; painful, fissured, corrugated tongue.
Vitamin C	Gingivitis, bleeding gingivae, loosening of teeth.
Vitamin D	Marginal gingivitis, alveolar bone resorption.
Endocrine Diseases	
(a) Pituitary	
(1) Hyperpituitarism (acromegaly)	Heavy, thickened, furrowed tongue, overgrowth of alveolar process with wide spacing of teeth, protrusion of lower jaw, thickening and eversion of lower lip.
(2) Hypopituitarism	Irregular alignment and crowding of teeth. Incomplete root formation.
(b) Thyroid	
(1) Hyperthryoidism	Increased flow of saliva.
(2) Hypothyroidism	Thickening of tongue, macroglossia, hypertrophy and puffiness of gingivae, lusterless enamel about necks of teeth, caries.
(c) Adrenals	
(1) Hypoadrenalism — (Addison's Disease)	Pigmentation of oral mucosa: bluish black or bronzing, bleeding gingivae.
(d) Pregnancy, menstrual, dyscrasias, menopause	Gingival changes, glossitis, dryness of the mouth.
Diseases of the Hemopoetic System	
(a) Anemias	
(1) Pernicious anemia	Bright red, smooth tongue with characteristic gelatinous surface. Greenish-white tint of soft palate.
(2) Plummer-Vinson Syndrome (microcytic anemia and vitamin deficiency)	Glossitis, dysphagia, lips thin and inelastic.
(3) Iron deficiency anemia	Pallor.
(b) Thrombocytopenic Purpura	Purpuric lesions on buccal mucosa, hard palate, gingivae.
(c) Polycythemia vera	Deep cherry-red color of oral mucosa, swollen, purple tongue.
(d) Agranulocytosis	Angina and erosions of soft palate, gingivitis with pseudo-membranes.
(e) Leukemias and other lymphoblastomas	Non-specific type of lesions on tongue, buccal, and pharyngeal mucosa.
Psychoneurotic disturbances	Glossodynia.
Gastro-intestinal diseases	
(a) Ulcerative colitis	Aphthous lesions.
(b) Generalized intestinal polyposis	Pigmented lesions on lips and buccal mucosa.
Other Systemic Diseases	
(a) Acanthosis nigricans (juvenile and adult types)	Condylomatous lesions on tongue, buccal mucosa and angles of mouth; thickening of buccal mucosa.

(IV) Exclusive Dermatologic Entities of Known or Unknown Origin	*Oral Manifestations*
Pigmentations	
(a) Vitiligo	Involvement of vermilion border of lips.
(b) Pinta	Depigmented and hyperpigmented lesions on lips and buccal mucosa.
Eczematous Dermatitides (allergic contact-type)	Erythema, edema, vesiculation. Contactants: cosmetics used for the teeth and mouth, dentures, fillings, troches, etc.
Papulo-squamous Diseases — lichen planus, pityriasis rosea, psoriasis.	Classical lesions of L.P. scaly element lacking, however. Denuded patches in P.R.
Bullous Diseases—pemphigus, dermatitis, herpetiformis, erythema multiforme exudativum (Hebra).	Characteristic bullae and erosions.
Benign newgrowths	Papillomas—tongue, gums, lips. Nevus pigmentosus—lips. Fibromas—tongue and gums. Keloids—tongue. Neurofibromas—buccal mucosa and tongue. Lipomas—dorsal surface of tongue. Angiomas, lymphangiomas, and hemangio-lymphangioma.
Precanceroses	
(a) Leukoplakia	Definitive entity.
(b) Erythroplasia of Queyrat; Bowen's	Shiny, glistening plague. May occur on lips and tongue. Rare.
(c) Xeroderma pigmentosum	Vermilion border of lips.
(d) Roentgen ray dermatitis	Lips.
(e) Arsenical keratoses and senile keratoses	Lips.
Malignant neoplasms	Basal cell carcinomas—extension from cutaneous surfaces. Squamous cell carcinomas—lips and mouth. Adenocarcinoma, hemangio-endothelioma, metastatic carcinoma, myoblastomas, etc.

It is apparent that some oral manifestations represent entities limited to the buccal mucosa and the skin. However, the majority of the oral lesions reflect, in a similar fashion as the skin, in identical or varied morphological aspects the pathological changes in other organ systems. It would be impossible to discuss all the aforementioned conditions and we have therefore presented a group of kodachromes as representative of the diseases listed. However an example from categories III and IV will be cited as a means of developing our thesis of interrelationship of skin and oral mucosa, and of skin, oral mucosa and internal disease.

Skin and mouth lesions in association with ulcerative colitis:—one of us (M.H.S.)[1] recently reported the skin manifestations associated with or complicating ulcerative colitis. The skin lesions were classified in the following morphologico-pathologic pattern with known or presumptive causal mechanisms.

In this study 34 per cent of the 189 individual patients reviewed had one or more skin manifestations and approximately 30 per cent of the lesions were in the buccal cavity. This high incidence of skin lesions found in association with ulcerative colitis has suggested an interrelationship previously not considered and the following theoretical concepts were projected:

TABLE 1.

	No. Cases	*Per Cent*	*Presumptive etiology*
(1) Aphthae and other mouth lesions	28	app. 30	1) allergic 2) viral 3) psychosomatic
(2) Ulcerations and pyodermas	22	app. 24	1) bacterial
(3) Urticaria (inc. palmar erythema)	16	app. 17	1) allergic 2) psychosomatic
(4) Erythema nodosum-like (inc. petechiae)	11	app. 12	1) allergic 2) toxic

TABLE 2.

The mouth lesions reported were as follows:

(1) Aphthae	14
(2) Other mouth lesions (varying degrees of glossitis, stomatitis, gingivitis)	14

The schematic arrangement of the skin lesions (table 1) opens an avenue to correlate the etiology and pathology of ulcerative colitis with the etiology and pathology of the associated skin entities. It is significant that the causal mechanisms attributed to the production of the dermatological entities; namely, allergic, toxic, bacterial, viral, and psychosomatic are similar to those agents proposed in the etiology of ulcerative colitis. It is also significant that the histologic picture of the different skin conditions at times parallels the histologic manifestations of various stages of ulcerative colitis. With such a relationship, one speculates whether the skin lesion is produced by the same mechanism, whether there is a sequential relationship of the viral and other types of infection, or whether the skin merely reflects the altered process in a primary focus in the colon.

In this study we followed a group of cases of ulcerative colitis showing the various mouth lesions and a group complicated by erythema nodosum. The aphthous lesions and the erythema nodosum preceded by 24 hours the acute attack of ulcerative colitis or became manifest 24 to 48 hours later. These skin lesions involuted with control of the symptoms of the colitis. This time sequence may offer a clue to the interrelationship of the behavior of the skin and the behavior of the colonic tract. Also, if Rowe's theory of food allergy is correct, the relationship of aphthous stomatitis and ulcerative colitis is indeed significant, for herpetic mouth lesions due to food allergy are not infrequently observed. Similarly, if viral etiology is accepted, one could imply that the virus is responsible for both conditions for viral etiology has been proven for primary herpetic gingivo-stomatitis. If the virus infection is solely in the colon, this primary focus could also incite, probably by an allergic mechanism, a second wave of infectious manifestations to produce aphthous lesions or erythema nodosum. In considering some agent like a leukotactic irritant or a circulating toxin, the same substance which is responsible for the colon lesions may also be responsible for the aphthous lesions. Possibly these agents by an altered sensitivity mechanism could produce petechiae and the lesions of erythema nodosum. The ulcerative skin lesions may result from the nutritional deficit suffered by these patients. These problems are posed as fields for speculation and perhaps further investigation. This is an example wherein the skin, and particularly the buccal mucosa, offers an opportunity to shed light on the behavior of a systemic disease such as ulcerative colitis.

Contact stomatitis is a well established entity. It has been reported repeatedly from plastic

dentures, denture creams, tooth pastes, mouth washes, amalgam fillings, essential oils, antiseptics, chewing of poison ivy leaves, medicated lozenges and even from foods. While most of these are based on true allergic sensitization of the oral mucous membrane, some result from a primary irritant action. Having accepted the existence of this entity we are confronted with certain peculiarities of its behavior.

There is an extensive and well authenticated literature regarding allergic cheilitis[2] which has been caused by lipsticks (most commonly the dye), tooth pastes, mouth washes, lozenges, dental plates, amalgam fillings, etc., readily confirmed by patch testing the lips and frequently the skin. Particularly noteworthy is the frequent absence of an associated stomatitis in these case reports, even though the offending agent is primarily intraoral in its location, such as dentures, fillings, tooth pastes, mouth washes, lozenges, etc. Of interest too, is the relatively infrequent involvement of the adjacent cutaneous areas except when oral mucous membrane sensitization can be demonstrated by patch testing.[3] The apparent rarity of contact stomatitis as compared with contact dermatitis is therefore noteworthy. Why, for example, can a patient with known contact sensitization of the skin to benzocaine often dissolve a troche containing this substance in his mouth without developing a corresponding stomatitis? Why do not all those patients showing a positive patch test to mercury on their skin react to the mercury in their amalgam fillings? On the other hand, why are patch tests on the skin to an acrylic resin so frequently negative when the use of a denture made of that material results in a prompt flare of the stomatitis?

The following are some of the suggestions offered to explain this relative infrequency of contact stomatitis: (1) the contact with the excitant is brief, (2) the agent is rapidly diluted by the saliva, and (3) the rapid dispersion of the irritant when it is absorbed keeps it away from the epidermal cells. This latter factor is dependent on the sparseness of a corneous layer, the thin epidermal layer and the extensive vascularization. Goldman and Goldman[3] conclude that it is the time factor of contact of the agent with the mucous membrane which seems to be the most likely reason for failure of sensitization in the sensitive individual. That in patients with a high degree of cutaneous sensitization, contact stomatitis does result inspite of this brief exposure to the agent. Goldman and Farrington[4] lend support to this assumption when they showed that 4 out of 16 patients with eczematous contact-type dermatitis due to penicillin which gave positive patch test reactions on the oral mucous membrane, also manifested facial dermatitis.

The following are some of the various methods of contact testing of the oral mucous membrane which have been used by Goldman:[5] (1) direct application by painting on the mucosa with a cotton-tipped applicator; (2) cotton roll, soaked with the solution and held under the lip; (3) fastening a thin bit of cotton to a denture or partial denture with collodion and then applying the agent to the cotton; (4) usage test, use of the material and then examination for a flare. This latter method is currently the most popular one in corroborating a diagnosis. In the past 10 years, investigators have been able to demonstrate that allergic tests can be performed on the mucosa of the mouth in the same manner as the skin. Goldman and Goldman[3] devised a standardized contact testing method employing a rubber suction cup. Farrington later modified this, using a rubber finger cot.[6] While the optimum time of exposure has not yet been worked out, it now varies from 20 minutes to 24 hours, the use of these more nearly standardized methods have opened the way for the systematic study of the problem of contact stomatitis. It is the hope of the authors that the dentist and the dermatologist will cooperate in a program of concurrent mucous membrane and cutaneous patch testing of all patients with contact type stomatitis.

To supplement the picture of dermatologic-dental relationships we have also included the following subjects:

Occupational Dermatoses of Dentists: Orland,[7] in reviewing the occupational hazards to which the dentist is exposed, enumerated the

following conditions: Roentgen ray dermatitis of the fingers when the dental x-ray films are held in position by the dentists; dermal hypersensitivity to formaldehyde, certain essential oils, methyl methacrylate monomer, x-ray developer, mercury and local anesthetics; irritant contact dermatitis due to soaps; transmissible infectious diseases as syphilis; and traumatic injuries due to fillings, calcretions, etc.

The council on Dental Therapeutics has conducted a survey to determine the prevalence of occupational dermatitis among dentists. The following statistics were made available.[8]

CASES OF OCCUPATIONAL DERMATITIS AMONG DENTISTS, REPORTED TO THE COUNCIL ON DENTAL THERAPEUTICS PRIOR TO OCT. 1, 1948.

Cause	*Number*
Local anesthetics	358
Unknown or not stated	75
Miscellaneous drugs and chemicals	70
Soap, water or frequent hand washing	50
Acrylics	35
Miscellaneous dental materials	32
Butacaine ("Butyn")	28
Tetracaine ("Pontocaine")	20
X-ray solutions	14
Benzocaine	13
Formaldehyde	11
Rubber or latex	11
Phenol	9
Benzalkonium ("Zephiran") chloride	8

It is evident that of the contactants, the local anesthetics are the chief offenders. The results of a questionnaire indicated that over 5 per cent of all Naval Dental Officers believe that they have had, or have procaine dermatitis. Sometime ago, one of us (M.H.S.) drew up a protocol to investigate this problem along the following lines: (1) protective measures employed by the dentist and their relative effectiveness; and (2) to expand on previous studies of the specificity of procaine and to determine the efficacy and sensitivity index of substitute anesthetic agents.

Occupational Stigmata of the Mouth and Teeth: With the present day distribution of workers into all conceivable fields of industry and labor, it is not surprising that the industrial dermatologist concerns himself not only with the affections of the skin incident to the patient's occupation but also must concentrate upon the various stigmata which involve the mucous membranes of the mouth, nose and eyes. Inasmuch as the dentist's interest is also primarily focused upon dental and oral mucosal changes it behooves both groups of specialists to familiarize themselves thoroughly with the pathognomonic and non-specific alterations which occur in the mouth and teeth as a result of occupational efforts and hazards. Occupational diseases may involve dental and oral structures to produce characteristic lesions and stigmata. The latter group of telltale markings may be divided into (a) odors, (b) stigmata of the teeth, and (c) stigmata of the oral mucosa. The following examples are illustrative:

(a) Odors: C. P. McCord in a talk before the American Association of Industrial Dentists said: "Dentists function in a world of odors, many of which are obviously unrelated to occupation. On occasion, breath odor discloses exposure to harmful agents, but in themselves furnish no real proof of actual disease. Most of the gases and vapors that enter the body through the lungs are also eliminated by the lungs. Of possible significance is the garlic-like odor among workers in arsenic, selenium, cyanides, and some higher alcohols as allyl. The possibilities are legion." Other characteristic odors which may be associated with occupational exposure are: bitter-almond odor in workers exposed to hydrocyanic acid compounds, the violet-like odor in handlers of oil of turpentine, the sweetish-metallic odor in workers showing chronic lead poisoning, the peculiar metallic fetor of mercurialism, etc.

(b) Stigmata of the teeth: teeth are frequently used in the performance of various types of work as well as being exposed to a diverse variety of noxious agents in industry. As a result of the repeated traumatic experience the teeth may show varying degrees and types of localized defects—often distinctly characteristic of the patient's form of employment, such as:

(1) Abrasions of the teeth and calculus formation may occur after exposure to dust encountered by polishers and blasters, abrasive powder workers and stonecutters.

(a) Localized abrasions of the teeth among garment workers, carpenters, cobblers, upholsterers, (tack spitter's teeth) due to foreign bodies in the mouth (nails, pins, etc.). The most characteristic stigma is the tack spitter's teeth which resemble a hacksaw.

(b) Bobby-pin destruction of the incisal edges of the upper anterior teeth among beauticians.

(c) Teeth deformities and abrasions among musicians (wind instruments), glass blowers, brush-bristle biters and seamstresses.

(2) Decalcification of teeth as a result of fume inhalations among acid dippers.

(3) Calculus, periodontitis and caries due to flour and sugar exposure among bakery workers; caries from sugar among candy workers.

(c) Stigmata of the oral mucosa: The oral mucosa itself usually manifests the vast majority of stigmata incident to occupational irritants and sensitizing agents. Mucous membrane irritation can be produced by deposits from the dust of asbestos, cadmium, chromium, cotton, cereals, powdered dyes, explosives, flour, grain, manganese, pollen, tobacco, wool, and all metallic dusts. It will result from contact with organic dusts from brooms, carpets, cotton, furs, hemp, rags, etc. All these aforementioned ingredients may act as chemical or mechanical irritants on the mucosa of the mouth. The lesions themselves may be erythematous, hemorrhagic, exudative, pseudomembranous, membranous, erosive or ulcerative in character.

In tabulated form the major offenders may be listed as:

(I) Chemical agents

a. Inorganic

(1) copper, brass, bronze, iron and nickel—green staining of teeth, pigmentation of gingivae and gingivostomatitis.

(2) lead—lead line (blue-black pigmentation) on the gums and gingivostomatitis.

(3) arsenic and bismuth—necrosis of bone, blue pigmentation of gums and oral mucosa—gingivostomatitis.

(4) chromium and chromic acid—orange staining of teeth, necrosis of bone and ulceration of oral tissues.

(5) halogens (Br, Cl, Fl, I)—discolorations and stains of the gingivae and oral mucosa.

(6) mercury—gingivostomatitis and salivation with metallic taste in mouth.

(7) phosphorous—gingivostomatitis, ulcerations of oral tissues.

(8) silver — (argyria) — violet line with metallic luster at gingival margins.

(9) inorganic acids—(sulfuric, nitric, hydrochloric)—bleeding stomatitis and decalcification of enamel and dentin.

(10) gases—(ammonia, bromine, chlorine) —stomatitis.

(11) inorganic dusts — staining abrasive gingivitis, bleeding, calculus.

b. Organic Agents

(1) Mother of Pearl—dust of this material in the manufacture of pearl buttons frequently produces painful acute gingivitis accompanied by fever.

(2) nitrobenzine—this chemical is used in the manufacture of aniline dyes and explosives; it is also used under the name of "oil or mirbane" or "oil of bitter almonds" in the manufacture of perfumes, liqueurs and confectionery. Oral manifestations consist of a greenish-blue discoloration of the mucous membranes, and sometimes a blue-black line on the gums.

(3) acrolein, amylacetate, cresol—stomatitis.

(4) benzol—hemorrhage from gingivae, stomatitis, blue coloration of lips.

(5) tar—stomatitis, malignancies of lips and mucosae.

(6) wine and liquor—anesthesia and paresthesia of tongue.

(II) Physical Agents

(1) atmospheric pressure changes.
increase (divers, caisson workers)—bleeding from gingivae.
decrease (aviators) — bleeding from gingivae.

(2) radium and x-ray—gingivitis, periodontitis, osteomyelitis and necrosis, xerostomia.

(III) Mechanical irritation

(1) ecchymosis and erosion of palate—incident to professional oral coitus.

Cosmetics for the Teeth and Mouth: Although the history of cosmetics dates back to

antiquity, the preparations advocated for the mouth and teeth are recent innovations. The popularity of the latter items has become so widespread—by appealing but untruthful claims in advertising and by the exploitation of supposed utilitarian functions—that no home medicine chest will lack some form of dentifrice or mouth wash. The value of these products is more illusory than real and infrequently untoward effects resulting from their use may be observed by the dentist and the dermatologist. The untoward reactions usually are of a local nature and depend upon irritant or allergic mechanisms. Systemic reactions due to absorption of various chemicals through the mucous membrane may also occur.

1. *Dentifrices:* the function of these products is to cleanse the surface of the teeth. Advertising, however, suggests alleged functions that they will sweeten the breath, prevent dental caries, sterilize the mouth, preserve receding gums, etc.

The current trent is to incorporate special constituents in many dentifrices for supposed medicinal effects. As a rule these ingredients are useless and at times may be harmful. Time and usage have debunked the alkalizing effects of milk of magnesia and the ineffectiveness of a host of germicides. The latter, in some cases, have produced deleterious effects because of allergic reactions. The claims of incorporated oxygenated products as sodium perborate and magnesium peroxide to enhance the whitening effect of dentifrices and to assist the removal of stains on the teeth have never been scientifically substantiated. The value of ammoniated dentifrices containing urea and amonium salts as preparations which will prevent dental caries by neutralizing decay-producing acids has not been conclusively proved by control studies. The promotion of chlorophyll as the dentifrice panacea has depended more on advertising copy writers than on verification in the laboratory. The most recent development in dentifrices has been the anti-enzyme dentifrice. Regarding the value of the aforementioned constituents, it suffices to mention the report of clinical work by a tooth-paste manufacturer which tended to prove that the main factor in preventing tooth decay is the simple brushing of the teeth immediately after eating. Probably it would be wiser if the dentifrice manufacturers return to selling people on brushing the teeth so that they will look clean and sparkling, rather than promote anti-caries panaceas.

The use of antibiotics as penicillin in dentifrices is mentioned only to be condemned . . . these drugs have no place in such preparations unless prescribed for a specific purpose by the physician or dentist. Although preliminary reports of the effectiveness of penicillin dentifrice on tooth decay in children seemed promising, subsequent studies did not prove statistically significant. Not only is the value of the antibiotic dentifrice unproved but its very use can produce alterations in the bacterial flora of the mouth to permit growth of monilia and penicillin resistant organisms. Also, the hazard of percutaneous absorption with subsequent drug sensitization must be considered, a not infrequent finding.

Allergic reactions to the various constituents can produce pictures of stomatitis, glossitis, cheilitis, perleche, or circumoral dermatitis. Patch tests using the ingredients of the dentifrice will establish the specific causative agent thereby allowing for a change in the dentifrice without harm to the patient. Recently, such ingredients as dichlorophene (G4) and cinnamon oil in ammoniated dentifrices have been reported as offenders.

2) *Mouth Washes:* Advertising ballyhoo dealing with halitosis opened an avenue to exploit the sale of this category of borderline medicinal-beauty preparations. The functions attributed to these products are antiseptic as the medicinal element, and a refreshing clean odor to the mouth, as the cosmetic element.

The antiseptics usually incorporated in such preparations include phenol, thymol, tannic acid, chlorinated cresols and thymols, esters of hydroxybenzoic acid, etc. Flavoring is made possible by blends of essential oils as spearmint, anise, cinnamon, lavender, myrrh, eugenol, clove, etc. Currently, chlorophyll has been advocated as the mouth wash panacea.

It is neither possible to disinfect the mouth or throat by a mouthwash or gargle nor is it

advisable to alter the normal bacterial pabulum of the mouth tissues by these commercial mouthwashes. The efficacy of these products has not been scientifically confirmed and in a study by the Food and Drug Administration most of the important brands on the market were toxic to the mouth tissue.

Despite reports that antiseptic mouthwashes in conjunction with dentifrices will correct halitosis, such claims have no scientific credence, for it is an established fact that bad breath usually results from metabolic and digestive disorders outside of the oral cavity. It is apparent, therefore, that the elimination of this complaint will depend upon the correction of the basic disorders. The characteristic unpleasant breath of habitual smokers is not corrected by these preparations and even the temporary unpleasant breath from eating garlic or onion is not overcome by any mouth wash. Indubitably, dental caries and pyorrhea are productive of bad breath; however, a far more practical approach is the elimination of the underlying pathology than the disguise by a mouthwash.

Reactions resulting from the use of these mouth preparations may be of an irritant, toxic nature or that of sensitization.

Dryness of the Mouth: Dryness of the mouth or xerostomia is a symptom sufficiently common in occurrence to warrant a careful study of its nature by both the dentist as well as the dermatologist. The intensity of the discomfort produced as well as the pathologic changes in the oral tissues are, of course, dependent upon the degree of involvement. Interestingly, the symptom of xerostomia per se is not incompatible with reasonably good general health unless the underlying mechanism responsible for the dryness is of a debilitating systemic nature. From this, it is immediately apparent that xerostomia does not represent a single etiologic and clinical entity but rather expresses a similar symptom pattern due to a multiplicity of causes, both local and systemic. Since the purpose of this review is to correlate medical and dental interrelationships and since proper therapy is founded upon exact etiologic evaluation, it follows that a tabulation of major causative mechanisms assumes a position of primary importance.

Local causes

A. Temporary

1. Mouth breathing, excessive speaking and normal emotional reactions.

2. Mechanical or obstructive causes: salivary gland tumors, obstruction of salivary ducts (stone), acute suppurative infection or infiltration—tuberculosis, syphilis, actinomycosis, etc.).

B. Permanent

1. Congenital hypoplasia or absence of salivary gland.

2. Physiologic—atrophic changes in salivary glands due to advanced age.

3. Physical agents — x-ray and radium damage.

4. Surgical operation with loss of gland substance.

Systemic Causes

A. Temporary

1. Drugs and toxic agents—atropine, opium derivatives, ergotamine and organic poisons.

2. Chemical disturbances in conduction or reception of nerve impulses to salivary glands.

3. Abnormal emotional states, neuropsychiatric disorders and psychoses can produce prolonged salivary suppression.

4. Dehydration—through hemorrhage, hyperhidrosis, diarrhea, vomiting, polyuria and acute febrile diseases.

5. Dietary and vitamin deficiencies as with vitamin A deficiency, iron deficiency anemias.

B. Permanent

1. Organic nervous system lesions—inadequate secretory stimulation.

2. Endocrine diseases—diabetes mellitus or insipidus.

3. Idiopathic—Sjögren's Syndrome: keratoconjunctivitis sicca, laryngopharyngitis sicca, and enlargement of the parotid glands.

Fluorescence of the Mouth: Fluorescence of the mouth is a subject which has created a greal deal of interest from a medicodental standpoint because of its curious nature as well as its possible diagnostic significance in relation to nutritional deficiency states. From the outset a distinction must be drawn between the beautiful red fluorescence of the teeth in congenital porphyrinuria of children when irradiated with

the Wood's light and the normal phenomenon of fluorescence apparent in the mouths of most healthy adults. In the latter instance, the substance causing the fluorescence was found to be coproporphyrin on spectroscopic study (Van den Bergh). As to the origin of porphyrins in the mouth, early speculative investigation considered bacterial mouth flora, salivary content of porphyrins and components of the food intake with associated bacterial action. However, today the opinion is quite definitely accepted that the porphyrins of the mouth result from bacterial synthesis. It is yet to be determined which specific groups of bacterial flora are the responsible productive agents. The phenomenon of fluorescence is elicited simply by examination of the mouth with the aid of the Wood's filter and yields a reddish-orange fluorescence on the dorsum of the tongue and sometimes on the teeth.

Most healthy adults demonstrate this phenomenon to a variable degree and intensity. In the majority of instances the posterior ½-¾ of the dorsum of the tongue shows the fluorescent feature with freedom of the tip and margins. However, about 20 per cent of healthy individuals exhibit fluorescence of the whole tongue. On examination with the naked eye, the fluorescence appears to be coalescent but on closer inspection through a magnifying lens, it is seen to be composed of fluorescent puncta corresponding to the filiform papillae. The significance of this observation becomes apparent when one considers the prerequisites necessary for the exhibition of fluorescence, namely:

(1) The presence of porphyrin producing bacteria.

(2) The presence of normal papillae, since a smooth tongue does not as a rule manifest the fluorescent feature.

In 1944 Costello and Luttenberger[9] attributed the absence of the normal fluorescence to vitamin B deficiency and in 1947 Hagerman and Hirschfeld[10] in an exhaustive study confirmed this observation and further noted that vitamin B preparations, especially those containing pantothenic acid, usually restored the tongue to its normal fluorescent state. In 1951, Tomaszewski[11] reported an excellent study of the entire subject of tongue fluorescence on over 400 patients. He found no significant differences with regard to sex but he did note a diminishing degree of fluorescence with advancing years. Generally, the intensity of the process was definitely less in old people as compared with children and young adults. Special attention was paid to diseases which are associated with changes in the tongue. In certain diseases such as pernicious anemia, hypochromic anemia, sprue syndrome and vitamin B deficiencies there is a higher incidence of absent fluorescence.

Another phase of this problem dealt with observations regarding the effect of antibiotics on normally fluorescent tongues. Administration of penicillin troches, streptomycin troches, oral chloramphenicol capsules and troches and aureomycin capsules produced disappearance of fluorescence within a few days after administration and reappearance on cessation of medication. Interestingly, penicillin by intramuscular injection for one week did not abolish the normal fluorescence. The obvious conclusion to be drawn from this study is that the antibiotics destroy fluorescence by their action on mouth organisms.

The clinical significance of these assorted data suggests the possibility of applying the fluorescence test as a simple diagnostic method of avitaminosis. Unfortunately, however, absent fluorescence is not always associated with vitamin deficiency. Apparently, some heretofore undiscovered factors of a nutritional or metabolic nature play a significant role in destroying fluorescence in a small percentage of healthy and adequately nourished individuals. Until further investigation reveals the underyling basis of this absent fluorescence, no clinical significance can be attached to this phenomenon. However, it does suggest an interesting avenue of approach for future study and clarification. The application of the Wood's light fluorescence as a diagnostic aid in various dermatological and oral disease processes has already been intensely studied and put to practical use. Prominent among these problems is the differential diagnosis of leukoplakia and lichen planus of

the mouth. Under ordinary circumstances when the clinical attributes of these two entities are encountered in the mouth in the typical or characteristic case, there is no difficulty involved. However, many times a real diagnostic dilemma is encountered in their differentiation. Since the prognostic significance of these two disturbances is so vastly different, any diagnostic aid which serves to distinguish them assumes clinical importance. Here the Wood's light is of decided benefit since it reveals distinct white fluorescence of the lesions of leukoplakia whereas lichen planus will not demonstrate this characteristic. This diagnostic aid should precede any staining procedures, for differentiation, as Lugol's solution.

Another recent and important application of Wood's light fluorescence is in the diagnosis of ulcerated squamous cell carcinoma of the skin and mucous membranes. The subject has recently been studied and described in full detail by Ronchese and co-workers[12] who made clinical observations in over 50 cases at the Tumor Clinic of the Rhode Island Hospital. The lesions they studied were distributed over various portions of the general skin surface and oral mucosa. With the aid of the Wood's light a peculiar vivid reddish-orange fluorescence ("live-coal-red fluorescence") was elicited from necrotic, ulcerated surfaces of squamous cell epitheliomas in contrast with a uniform deep violet discoloration of other ulcerated surfaces (basal cell epithelioma, carbuncle, tubercular and syphilitic gumma, granuloma fungoides, pseudoepitheliomatous hyperplasia, and long standing inflammatory process). The value of this observation is, of course, self-evident in determining the nature of a necrotic, ulcerated lesion. Not only will it serve to suggest the proper diagnosis but will also help to rule out conflicting opinions offered by the other types of lesions.

Finally, although not pathognomonic of any distinct entity, the true extent of eruption of many dermatoses may be better discerned with this method; cutaneous and mucous membranes lesions which do not show definite color contrast with their background usually can be more distinctly delineated under the Wood's light, for example the lead line and bismuth line of metallic poisoning.

CONCLUSION

In this random discussion, many specific problems have been left untouched. Their omission does not indicate a lack of awareness of the multiple ramifications of medico-dental inter-relationships but rather a limitation of time and space. Primarily, this presentation was restricted to a threefold objective, to encompass a consideration of:

(1) Dermatologic disorders which show major oral components.

(2) Systemic disorders associated with both oral and cutaneous manifestations.

(3) Specifically selected problems of mutual interest and importance to both the dentist and the dermatologist, including: (a) occupational dermatoses of dentists, (b) occupational stigmata of the mouth and teeth, (c) cosmetics for the mouth and teeth, (d) fluorescence of the mouth and (e) dryness of the mouth.

This outline was discussed and illustrated with primary emphasis on clinical approach and application. If this paper has served to transcend the narrow boundaries of over-specialization and has helped to widen the horizon of medico-dental integration, then our aim has been achieved.

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RELATIONES DERMATO-DENTAL

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SUMMARIO IN INTERLINGUA

Le specialisation intra le campo del medicina e del dentisteria ha efficacemente resultate in promover nostre cognoscentias e in ameliorar le sanitate del homines. Infelicemente, un del ben-recognoscite disavantages del tendentias specialisante ha essite le satis stricte maniera in que le majoritate del specialistas ha concentrate lor attention super le restringitissime aspectos de lor specialitates individual. Le responsabilitate del dermatologo non es complite si ille se dedica exclusivemente al pelle. Su attention debe expander se usque a includer le regiones ubi le pelle se trova in correlation con altere perturbationes functional o organic e ubi ille pote associar con le pelle le datos clinic e laboratorial colligite in le examine de altere systemas organic. Nostre objectivo deberea esser superar le deficientias del specialisation per disveloppar un attitude integrate que se occupa del complete stato sanitari del patiente. Isto es un objectivo que nos pote attinger si specialistas in cata un del restringite campos de recerca o de practica clinic succede a coordinar lor separate fragmentos de cognoscentia pro le beneficio mutual del scientia e del homines.

Le importantia de iste programma es generalmente admittite, e il es interessante insister super le rolo del dentisteria in iste effortio cooperative. Le dentista associate con un bon servicio dermatologic non es le gerente de un clinica dental. In tanto que chirurgo-dentista ille debe considerar se como membro essential de un cooperativa tumorologic que include etiam le radiologo, le chirurgo plastic, le pathologo, e le dermatologo; in tanto que medico dental ille debe facer se parte del consilio medico-dermatologic que include le internista, le hematologo, le pathologo, le endocrinologo, le psychiatro, e le consultante periphero-vascular. In plus, problemas de investigation require non infrequentemente le cooperation del personal medico-dental. Un exemplo de coordination de problemas investigatori e clinic pote esser prendite ab le clinica dermato-allergiologic ubi le dentista ha le opportunitate de construer le apparatura requirite in tests a emplastro intra le cavitate oral. Le idea que nos tenta projicer es que limitar le consultation dental primarimente al rango de auxilio in le tractamento del patiente dermatologic es myope. Pro compler su mission intelligentemente, le dentista moderne deberea supplementar intra le quadro hic discutite su cognoscentias de infection, immunologia, physiologia, e nutrition. Il es in iste direction que le nove tendentias del medicina se manifesta, i.e. le tendentias que cerca a transformar nostre conception del medicina curative de plus in plus a in un conception de medicina preventive.

Le presente revista es un tentativa de coordinar e synthetisar le specialitates dermatologic e dental super le nivello clinic. Il esserea bon extender nostre presentation de maniera a includer relationes physiologic e physiochimic, sed un tal plano es nimis pretentiose pro le spatio restringite de iste articulo.

Nos presenta synopticamente le mechanismos causal que es responsabile pro le morbos dermatologic e lor correspondentias dental. Le relationes dental es discutite in duo categorias: (1) Le manifestationes oral del dermatoses—le morbos del pelle que exhibi major manifestationes oral—e (2) manifestationes dermal e oral que es associate con morbos systemic. Pro completar le presentation del relationes dermato-dental, le sequente themas es discutite: (1) Dermatoses professional de dentistas, (2) stigmas professional del bucca, (3) cosmeticos buccal e dental, (4) fluorescentia del bucca, e (5) siccitate del bucca.

AN IMPROVED ANTIBIOTIC-ANTIHISTAMINE COMPOUND FOR ROOT CANAL MEDICATION (D.C.P.)

GEORGE G. STEWART, A.B., D.D.S.*

INTRODUCTION

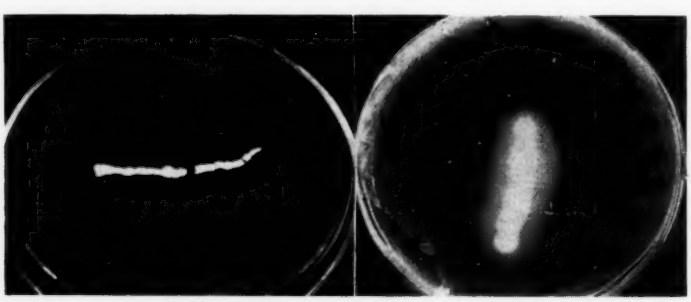
Improvement in chemotherapy, with emphasis on the antibiotics, has been marked in recent years. The story of the development of the antibiotics is common knowledge and will not be reviewed here. All of the better known compounds are now being used in treating dental and oral infections.

The usefulness of penicillin as a root canal medicament has been well established.[4, 6, 11, 12, 16, 18, 20, 22, 28] However, combinations of antibiotics have been found to be more effective in controlling the microbial life in the canal because of the variety of organisms that can be found there.[1, 23]

Streptomycin was among the first compounds added to penicillin to increase the antimicrobial spectrum.[5, 14, 17] This combination controlled most of the gram-positive and gram-negative organisms found in the root canal. It did not, however, control the yeast-like organism found in the canal.

Grossman[15] added bacitracin to the penicillin-streptomycin combination to destroy the enterococci, and sodium caprylate to destroy the yeasts. Seltzer and Bender[19] modified this combination and substituted chloromycetin for bacitracin. The mixtures suggested by these workers are very effective because they inhibit the growth of all the organisms commonly found in infected root canals.

However, the problem of penicillin sensitivity, although a minor factor in root canal therapy, must not be overlooked. The author has had two patients manifesting systemic (dermatologic) reactions. One case was reported elsewhere.[25] Because of this, an antibiotic-antihistamine compound for root canal medication was introduced.[25] The antihistamines protect the patient against allergic reactions and some have excellent antimicrobial activity as well.[8, 9, 24]



1A 1B

Fig. 1. A comparative study of the diffusibility and inhibitory action (1-A) P.C.P. (potassium penicillin G-chloromycetin-Perazil) in Neobase, to (1-B) D.C.P. (dibenzyl penicillin-chloromycetin-Perazil) in Xylocaine ointment base. The plates were seeded with Canolida albicans, Escherichia coli, and Streptococcus faecalis.

This paper will report on an improved antibiotic-antihistamine compound that has been used in the office of the author for more than two years. A portion of this study was presented at the Greater New York Dental Meeting in December, 1953.

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LABORATORY STUDIES

The details of the preliminary laboratory studies have been reported.[24, 25] However, Figure 1 does show the greater diffusibility of DCP as compared to PCP when similar conditions of testing were used. The organisms used were Candida albicans, Escherichia coli and Streptococcus faecalis. These organisms were obtained from pure stock cultures and grown separately in brain heart infusion broth (Difco) for 24 hours at 37° C. They were then subcultured to insure vigorous growth. Just before plating, the three tubes of organisms were combined and were mixed vigorously by shaking for five minutes. With a sterile pipette, 0.2 cc was withdrawn and discharged on to the surface of a petri plate which had been covered with 10 cc of solidified blood agar base. With the aid of an "L" shaped glass rod, the microbial suspension was evenly distributed over the surface of the agar.

Small amounts of the DCP and PCP cream were ejected on to the agar, and the plates were then incubated for 24 hours. The photographs show the results of the study.

Small portions of the clear area were removed with a sterile loop and innoculated into tubes containing 10 cc of sterile brain heart infusion broth. The tubes failed to show growth after they had been incubated at 37° C for one week.

PREPARATION OF THE D.C.P. CREAM

Formula:

Dibenzylethylenediamine-Penicillin (Bicillin)	300,000 units
Chloromycetin (crystalline)	125 milligrams
Perazil (antihistamine)	100 milligrams
Propylene glycol	0.25 c.c.
Xylocaine Ointment 5%	0.25 c.c.

Preparation:

Place 4 Perazil tablets (50 mg. each) in a sterile mortar, as used for mixing amalgam, and triturate to a fine powder. Add to this, the contents of a 250 mg. capsule of Chloromycetin and triturate further. The Bicillin is available in 1 c.c. disposable carpules with needle attached. Each c.c. contains 600,000 Units of Dibenzylethylenediamine-Penicillin. The Penicillin is added and a paste-like mass is produced by mixing it with the Perazil and Chloromycetin. To bring the mass to a cream consistency, 0.5 c.c. of 5% Xylocaine Ointment is added and 0.5 c.c. of Propylene glycol (approx. 7 drops) is added. The mixing is continued until a smooth creamy consistency is

2A 2B 2C

Fig. 2. An acute alveolar abscess—showing repair of periapical tissue when treated as described. The time interval between 2-B and 2-C is six months.

obtained. If a thicker cream is desired, the Propylene glycol can be eliminated and 0.5 c.c. of 5% Xylocaine Ointment can be used for each 1.0 c.c. of total cream preparation.

The cream is stored in a 5 c.c. Luer-Lock syringe or in empty anesthetic carpules.

CLINICAL PROCEDURE

The clinical study of the first 100 teeth treated using D.C.P. cream as the root canal medicament is presented here.

Teeth containing vital pulps were not selected for this study.

The method of treatment is essentially that described by Grossman.[11] The only difference is more frequent irrigation. A 3 per cent hydrogen peroxide solution was reacted with a double strength sodium hypochlorite solution after each instrument was used in the canal. These solutions were used primarily to flush the canal of loosened debris. If the canal is not flushed, these particles may be forced to the apex where they can block the apical foramen or be forced through the foramen where they can initiate additional periapical irritation.

After adequate cleansing and enlargement of the canal, it was dried with sterile absorbent points and the D.C.P. cream was introduced. A Luer-Lock syringe with a blunt angulated needle was used to introduce the cream. The canal was filled to overflowing, after which a thin absorbent point was introduced to the apex several times to insure thorough filling of the canal. The point was then discarded and a small pledget of sterile cotton was placed in the pulp chamber. Gutta-percha was then used as a plunger to gently compress the cream to the apex. Pressure was discontinued when the patient became aware of a feeling of "fullness" in the tooth. If the pressure caused discomfort, the cotton and gutta-percha were removed. They were replaced with another pledget of dry sterile cotton and gutta-percha. This procedure generally brought comfort to the patient. A second seal of cement was then placed over the gutta-percha, for greater protection against leakage.

SECOND TREATMENT

When the patient returned, the tooth was again isolated by means of a rubber dam and the entire area swabbed with untinted tincture of Metaphen 1:200, followed with 70 per cent alcohol.

After removing the double seal, the canal walls were dried with several sterile absorbent points. The test point was then inserted to the apex and permitted to remain there for one minute. It was then removed, placed in a tube of brain-heart infusion broth (Difco), and incubated at 37° C. for one week.

During the early part of these studies, "Penases Ascites Medium" (Difco) was used for 24 cultures. These all failed to show growth. It was then decided to use the brain-heart infusion broth as the routine culture medium. After taking the culture, the canal was further enlarged when required. In those cases where the enlargement had been completed, the walls of the canal were freshened with the final size instrument used in the original cleaning. The canal was again irrigated and dried as

TABLE I. AN ANALYSIS OF 100 TEETH TREATED WITH D.C.P.

Male	*Female*	*Age Distribution of Patients*						
47	53	0-9	10-19	20-29	30-39	40-49	50-59	60 over
		0	11	16	19	22	20	12

Anterior	*Premolar*	*Molar*	*Periapical Area*		*Cultures Required*	
			Yes	No	One	Two
48	26	26	83	17	94	6

described previously, before the fresh medication was introduced in the canal.

At the next visit, if the culture tube was free of growth and the tooth clinically comfortable, the root canal was filled. Prior to filling, however, the canal walls were again freshened and irrigated as described.

RESULTS

A statistical evaluation of the teeth treated is shown in Table I.

DISCUSSION

The reader will note that great emphasis has been placed on the thorough cleansing and enlargement of the root canal. This procedure is probably the most important phase of therapy. Grossman[10,11] and others have stressed this in their writings. Auerbach[2] has presented experimental evidence to prove this point.

The use of medication, however, is also important because, all the organisms in the canal may not have been removed or destroyed via bio-mechanical preparations alone. Turkenkopf[26] has shown the diffusibility of a Polyantibiotic compound through tooth structure. He demonstrated how it could penetrate the dentinal tubulae and cause inhibition of microbial growth in a seeded petri plate.

Wach and Hauptfuerhrer[27] have also demonstrated the diffusibility of penicillin from the root canal through the dentinal structure. They used radio active penicillin in their experiments and thus confirm the work of Turkenkopf.

These workers have answered experimentally the problem of what happens to the bacteria that find their way into the dentinal tubulae of infected root canals.

The question of the validity of culture findings when concentrated mixtures of antibiotics are used in root canal therapy has been raised.[7] It is a reasonable question because trace amounts of the antibiotics can inhibit microbial growth. Neutralizing agents that could safely be used in the culture media are not known for all the antibiotics used.

The value of the culture, however, should not be underestimated. The growth free culture alone, may not tell us when the canal is ready for filling, but a positive culture should indicate when not to fill.

From the view point of the clinician, however, the picture is much brighter. If minute traces of antibiotic substances are capable of inhibiting microbial life, could we not then expect concentrated quantities of the substances to be even more effective? Clinical experience has shown this to be true.[4,5,13,14,15,16,18,19,22,25]

3A 3B 3C

Fig. 3. A "Granuloma" treated as described. The time interval between 3-B and 3-C is one year.

4A 4B 4C

Fig. 4. A chronic abscess treated as described. Note the marked degree of healing between 4B and 4C in less than six months.

In the preparation of the medication, Dibenzyl-Penicillin (Bicillin) was used. It is a very stable compound having a shelf life of almost one and one-half years. The 1 c.c. disposable carpules that it comes in, are convenient to use, and it does lend itself to a smoother, more freely flowing preparation.

The role of the penicillin in the preperation is primarily to control the growth of gram-positive organisms found in the root canal.

Chloromycetin was added to enhance[25] the activity of the penicillin and help control the growth of the gram-negative organisms. It will not tend to discolor the tooth structure as the yellow or orange type broad spectra antibiotics will.

Streptomycin could not be used because of incompatability with the antihistamine compounds.[25]

The addition of the antihistamine serves several purposes. As has been reported, these compounds can inhibit the growth of fungus and yeast-like[8,9,24] organisms. Some are also effective in inhibiting the growth of other microorganisms.[21,24]

These compounds may also help protect persons from possible allergic reactions to the antibiotics.[3,25]

The antihistamines are also known to have anesthetic properties which are here enhanced by the addition of xylocaine ointment which is used as the vehicle. This factor tends to give the patient more comfort following instrumentation.

The ingredients for the preparation of the D.C.P. cream are readily available and the preparation is quite simple. The cream will flow freely and not cake in the needle or canal. Since the base is water soluble it can be removed easily from the walls of the canal. The greater stability of the D.C.P. is very important to the general practitioner who only occasionally resorts to root canal therapy. The cream has been useful in other dental problems such as in dry sockets, badly infected periodontal pockets and under inflamed third molar flaps.

In view of the properties stated and the clinical results obtained it would seem that the D.C.P. cream is a useful adjunct to root canal therapy.

SUMMARY

1. Laboratory evidence is presented showing the activity of an improved antibiotic-antihistamine compound upon mixed microorganisms.

2. The method of preparation and administration of the D.C.P. cream has been presented.

3. The effectiveness of the compound is demonstrated.

4. The advantages of the improved compound have been presented.

5. Some of the cases treated have been under observation for more than two years.

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UN MELIORATE COMBINATION DE ANTIBIOTICO E ANTIHISTAMINA PRO LE MEDICATION DEL CANAL DEL RADICE DENTAL

Dr. chir. dent. George G. Stewart

SUMMARIO IN INTERLINGUA

Progressos chemotherapeutic—specialmente in le campo del antibioticos—ha essite marcate in recente annos. Le historia del disveloppamento antibiotic es generalmente cognoscite e non es discutite in iste articulo. Omne le melio-cognoscite compositos es nunc in uso in tractar appropriate infectiones dental e oral.

Le presente articulo sublinea le necessitate de allargar e deterger le canal del radice con grande exactitude si on vole assecurar un bon successo del therapia. Le medication es solo un adjuncto del tractamento. Antibioticos ha essite utilissime in neutralisar le crescentia del organismos que ha possibilemente supervivite in le canal del radice post le plus caute detergimento. Proque le organismos incontrate in inficite canales del radice pote representar varie formas — gram-positive, gram-negative, e etiam saccharomycetic — combinationes de plure antibioticos se ha provate plus efficace in neutralisar los que un sol antibiotico non-combinate.

Le crema hic describite sub le nomine de "DCP" consiste del sequente componentes: Dibenzylo-penicillina (bicillina), un stabilissime preparato que servi super toto a controlar le crescentia del organismos gram-positive; Chlormycetina que es capace a promover le activitate del penicillina e a promover le controlo del crescentia del organismos gram-negative; e Perazil®, un composito antihistaminic que es capace a inhibir le crescentia de fungos e saccharomycetes como etiam de altere micro-organismos. Il es possibile que iste ultime possede in plus le virtute de reducer o prevenir reactiones allergic in personas sensibile al atere componentes del crema.

Le supra-describite crema esseva usate in le tractamento de 100 dentes. Post un sol tractamento illo resultava in culturas sin crescentia in 94 pro cento del casos. Excellente curationes esseva etiam notate, mesmo in casos de extense affectiones pathologic del area periapical.

CARDIOVASCULAR DISEASE AS IT AFFECTS DENTISTRY*

CLARENCE DENTON, M.D.†

The announced subject — "Cardiovascular Disease" — is too broad to be covered as a whole, nor would it have interest for you in any practical sense. Its relation to dentistry however, may be a more manageable and more pertinent topic for the members of your profession. Some of the overlapping problems in the two fields will be considered. Since I know little about dentistry, I shall divide the problems in a cardiologist's fashion: A) Determining the existence and nature of the cardiac disease. B) Knowing the particular problems associated with each disease. C) Knowing the means of solving the problems.

A. DETERMINING THE EXISTENCE AND NATURE OF THE CARDIAC DISEASE

A dentist may view the mouth as an organ with teeth that have many cavities. It is also a cavity with teeth and a tongue that can be used for talking. Making use of this apparatus is the first and simplest method of determining the existence of extradental disease in a patient. Ask him questions. These need not be many or complicated. They may even conceivably be omitted with those patients whose initial examimation reveals a need for only the filling of small cavities or cleaning of the teeth.

In all other patients put the question: "How is your general health?" or "Do you have any serious illness?" The latter perhaps is less likely to evoke a long series of neurotic complaints. This is more or less an introductory question, since either a positive or negative answer requires further probing, along these lines: Have you ever had diabetes, high blood pressure, a heart murmur or heart trouble? Have you ever had rheumatic fever or St. Vitus dance or swollen joints as a child? A negative answer to these questions and to one about drug reactions probably is a safe guide to proceed with the necessary dental work. If one of these questions has been answered affirmatively, it is not within the dentist's province to make a diagnosis or even to assume that the patient is a reasonable judge as to the significance of his condition. The next move is to find out exactly what the status of the disease is by talking to a physician.

A physician who has seen the patient within the last six months may be able to provide a diagnosis and an opinion as to the severity of the disease in that patient. If such is not the case, the patient should be asked to see his physician for an examination, so that the dentist may have an opinion that is currently worth something. There are few patients with serious heart disease who will resent such a request, especially if an explanation is made that his condition may increase the risk of the proposed dental procedure, unless adequate precautions are taken and that the dentist is not able to evaluate non-dental conditions.

The patient who goes to his physician should take a form from the dentist with a place to fill in the diagnosis and the physician's estimate as to whether the patient is in good enough condition to be up and around in his daily routine activities. It is better to ask for this sort of information rather than an opinion as to the patient's fitness for anesthesia or a specific surgical procedure. In a large group or in a hospital, where medical examinations for anesthesia or surgery may be routine, such bypassing of the essential question is not necessary, but too many physicians in private practice, who would be perfectly willing to permit a cardiac patient to be up and around, would be frightened at the thought of giving an okay for anesthesia for this same patient. In general, anyone who can carry out his daily routine of housework, or a

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job, or business can be given anesthesia safely, provided certain precautions applicable in the given disease are taken. A suitable form which the patient could bring to his physician for completion by the latter is as follows:

Dear Doctor:

This patient believes that he (she) may have some form of cardiovascular disease. Will you please examine him and let me know:

1) Diagnosis.
2) Your opinion as to ability to pursue normal daily activities.
3) Any other special factors in this case (e.g., Drug allergy, low pain threshold, emotional lability, cyanosis, cardiac irregularities, anticoagulant therapy, etc.)

I feel that it is necessary to make separate mention of cardiac irregularities under item three because often a physician will fail to mention such, especially premature beats, under the heading of diagnosis where this belongs. Such beats in a patient with heart disease may be a warning that a serious arrhythmia could occur at surgery. In general, diagnosis will be self-explanatory but I think that it might be well to point out that a diagnosis of neuro-circulatory asthenia or a nervous heart means essentially no organic heart disease.

B. PARTICULAR PROBLEMS ASSOCIATED WITH DISEASE

I) All forms of heart disease.
   a. Arrhythmias.
   b. Congestive failure.

II) Congenital and rheumatic heart disease.
   a. Subacute bacterial endocarditis.
   b. Cyanosis.
   c. Bleeding from anti-coagulant therapy.

III) Arteriosclerotic heart disease.
   a. Angina.
   b. Coronary thrombosis.
   c. Arrhythmias.

IV) Hypertensive Heart Disease.
   a. Hypotension.
   b. Increased hypertension and hemorrhage.

Normal patients as well as patients with heart disease may have episodes of abnormal cardiac rhythm or rate. Certain types however—the more serious ones—occur only in patients with damaged hearts, and all types are more common in patients with heart disease. The commonest varieties which may occur in normal or diseased hearts are ectopic beats, paroxysmal atrial tachycardia, and paroxysmal atrial fibrillation. Chronic atrial fibrillation and varying degrees of heart block are the commonest varieties seen in patients with heart disease. While the commonest cause of such arrhythmias is arteriosclerotic heart disease, none of them is limited to this disease and the risk of bringing on an attack during anesthesia or a surgical procedure, while present to a greater degree, in arteriosclerotic patients, is a possibility in any cardiac. A predisposing factor in the development of certain arrhythmias is increased irritability of the myocardium. Two things among others are important myocardial irritants—anoxia and epinephrine. Mention of these will suffice for the moment more will be said of them later. Any excess sympathetic stimulation is likewise a factor; pain and emotional disturbances associated with fear fall in this category. The other condition which may occur in any form of heart disease is congestive heart failure. This is an expression of decreased myocardial reserve—that will o' the wisp of the physiologist, so easy to talk about, so impossible to measure.

The heart function is simple. It is a pump. When it is functioning adequately, it permits an individual to perform any physical activity within range of his muscular skill and power. Such a patient may still have heart disease, but in the American Heart Association classification, he is a Class I cardiac. When there is a loss of reserve strength, so that ordinary exertion causes dyspnea, then we have a Class II patient. A still greater decrease in function, so that dyspnea occurs on less than ordinary exertion but not at rest, puts the patient in a Class III category. Finally, the Class IV patient is dyspneic at rest. The patient in class IV is not likely to come to your office, and in the hospital he is a candidate for only emergency dental

procedures, until his cardiac condition can be improved by the proper therapy.

If such is unsuccessful, the prognosis for life is probably not sufficient to warrant expensive and extensive procedures in the oral cavity, which the patient will not survive long enough to appreciate. The class I, and class II patient will certainly run no risk of cardiac decompensation as a result of any surgical procedure, and it is for this reason that the physician is asked to state his opinion about the patient's ability to carry on his daily activities. For, if the patient can do this with ease, he is no worse than class II. The class III patient may represent a distinct problem, although generally, with a little care and avoidance of long drawn out surgical procedures, this patient too can undergo dental surgery. Occasionally, an actual consultation between physician and dentist may be necessary and may go a long way to resolving the difficulties.

Congenital and rheumatic heart disease pose three separate problems, in addition to the arrhythmias and congestive failure. The first and most important of these is subacute bacterial endocarditis or endarteritis. This bacterial infection of the cardiovascular endothelium occurs, for practical purposes, only in those individuals with damaged or abnormal valves or abnormal communications between the heart chambers or major blood vessels. The path of infection is from a primary site, where bacteria enter the blood stream, to the endocardium. Such invasions of the blood stream are apparently common following many surgical procedures. 80% of patients had bacteremia following post-abortal uterine curettage. 33 of 80 urologic patients had positive blood cultures postoperatively, when they were negative preoperatively. 51 of 60 tonsillectomies had bacteremia, and 84 of 138 dental operations had post-surgical bacteremia. These sample figures from the literature should suffice to save a dentist from guilt feelings. The bacteremia is apparently quite transient because the high percentage quoted applies only in the first 15 minutes after surgery. In the vast majority of patients, apparently no tissues are harmed by this bacteremia. It is only in the patients with congenital abnormalities or rheumatic scarring of a valve that apparently a site of lesser resistance permits bacterial invasion, usually by an otherwise mildly pathogenic organism, the streptococcus viridans. The actual percent of cases having bacteremia that go on to develop bacterial endocarditis is not known. In the old literature it was said that 25 percent of patients who died as a result of rheumatic heart disease, did so from bacterial endocarditis. Hence, apparently an appreciable percentage of those with scarred valves run the risk of a serious illness secondary to the bacteremia. Of course, not all of the cases have been attributable to dentists, but the proper method of prevention is so cheap and easy and the proper method of treatment so prolonged and expensive that the dentist should never neglect to play his part in prevention.

The second problem that may present itself under this heading is cyanosis. This is merely a sign of an increased amount of reduced hemoglobin in the blood stream. It is not pathognomonic of congenital heart disease, nor is it present in all kinds of congenital heart disease, but its presence or potential presence should be made known to the dentist, so that he will be careful not to increase the degree of oxygen unsaturation. The risks of doing so are: 1. increased myocardial irritability with possible precipitation of an arrhythmia; and 2. decreased oxygen supply to all organs, but especially the brain, with the possibility of producing some serious neurologic deficit. It is well to remember two things about cyanosis: first, its apparent absence in a patient with congenital heart disease may merely mean that the amount of desaturated hemoglobin is not enough to be noticeable. Even with a normal hemoglobin, it may be impossible to see cyanosis with an oxygen saturation of 85%; yet if this patient were to struggle or for any reason to have an increased resistance in the pulmonary circuit, the degree of right-to-left shunt through his congenital defect might increase and he would then become visibly cyanotic, perhaps dangerously so. Second, its presence in a patient with some type of heart disease does not necessarily signify a congenital lesion. The cyanosis may

be peripheral in origin rather than central, due to congestive failure and stasis in the tissues, where more oxygen than normal is extracted from the blood. In these circumstances, the blood returning to the heart has a decreased oxygen saturation, but all of it goes through the lungs and becomes fully saturated, so that blood flowing from the heart to all organs has a normal oxygen content—a quite different state of affairs from that in cyanotic congenital heart disease.

The third item of importance is the frequent use of Dicumarol or similar products to prevent thrombo-embolic phenomena in heart disease. Chronic atrial fibrillation with mitral stenosis often results in clot formation in the atria; and emboli to the brain, kidney, extremities, lungs and other organs occur in 10 to 15 percent of all cases of mitral stenosis. Many physicians make use of anticoagulants to try to prevent such occurrences. It is of obvious importance to the dental surgeon to know if his patient has any bleeding tendency, be it due to disease or drugs, and it is for this reason that anticoagulant therapy is so specifically mentioned on the form suggested.

Arteriosclerotic heart disease is far-and-away the commonest form of heart disease in the United States today. As such, it is often present without any symptomatic manifestation, and in this form will give rise to an occasional unavoidable sudden death in a dental chair. Its major distinctive manifestation, when symptomatic, is pain, a pain ordinarily brought on by exertion, excitement or large meals, and relieved by rest. Such episodes are referred to as angina pectoris and are believed due to a temporary decrease in blood supply to the heart muscle, or an increase in its work load, or both. The actual pain producing factor is considered to be an oxygen-lack, relative to the work load. Longer lasting pain, unrelieved by rest, may represent so-called coronary insufficiency, or even a complete occlusion of a coronary artery. Since pain can produce a tachycardia, since struggling and fear increase the cardiac output and rate, since bleeding and hypotension can increase the heart rate, and since oxygen-lack directly affects the heart muscle, I think it should be apparent in what fashion the dentist may precipitate an attack of angina or coronary insufficiency, or even a myocardial infarction. As has been previously stated, this is the type of heart disease which is most subject to sudden arrhythmias, some of which may be fatal. Even a few ventricular ectopic beats may be the forerunner of a fatal ventricular tachycardia and fibrillation. Pain, fear, struggling, and hypoxia all lead to overstimulation of the sympathetic nervous system, with possible resultant alteration in the normal rhythm of the heart. One final danger which should also be mentioned here again is the possibility that the patient may be a bleeder, as the result of Dicumarol therapy, which is widely used in the long-term management of some cases of myocardial infarction. In the acute phase it is used while the patient is still in bed to prevent venous thrombosis and pulmonary embolism. In the ambulatory patient it has been used to prevent further propagation of the clot in the coronary artery (a very doubtful possibility) and to prevent mural thrombus formation at the site of the infarct with subsequent peripheral embolism. Again it is hardly necessary to point out the danger of possible bleeding in such a patient.

Perhaps the commonest single potentially serious illness in the United States today is hypertension. Certainly it ranks well up with peptic ulcer and is probably more frequent than diabetes. In most patients it is asymptomatic and compatible with many years of normal activity. Nevertheless in this group of patients, arteriosclerosis tends to occur earlier and more severely than in the normotensive population, and diabetes is more frequent. Hence all of the precautions mentioned under the heading of coronary arteriosclerosis may be applicable and it is worth while knowing if a hypertensive patient has this complication. In the absence of renal or cardiac involvement, surgical procedures are withstood well by the hypertensive patient. Two complications of surgery are to be avoided if possible. These are a marked rise or a considerable fall in blood pressure. Pain and struggling give rise to an elevation in vascular tension and this may be enough to lead to intracranial bleeding, either from a slightly arteriosclerotic vessel

or from the less common but by no means rare congenital aneurysms that may exist in the cerebral vessels, especially in the circle of Willis. Hypotension may result from blood loss, so-called neurogenic shock secondary to pain, or from a drug reaction. It may be of no consequence unless there is associated cerebral or coronary arteriosclerosis. In states of hypotension, particularly when previous hypertension had been necessary to sustain adequate blood flow to a portion of the myocardium or brain through narrowed sclerotic vessels, there may occur such a decrease in blood flow as to lead to a myocardial or cerebral infarct. There may have been a previous history of angina, or coronary thrombosis, or stroke to make one aware of the existence of significant arteriosclerosis, but this need not have been the case. Indeed, in the heart at least, there need not even be significant arteriosclerosis. A very large heart—often seen in hypertensives requires a greater blood supply than a normal sized heart. A considerable fall in blood pressure may lead to angina or myocardial infarction even without partial or complete vascular occlusion. There may always be a first attack, and it may occur in your dental chair, if reasonable precautions are not taken to maintain blood pressure, or at least to avoid situations that lead to hypotension.

C. THE METHODS OF HANDLING THE PROBLEMS

We now come to the question of what to do about the various difficulties which have been postulated. In discussing them, so as to avoid repetition, it is wise to consider the factors which the dentist can control. These are: 1. anxiety, 2. pain, 3. hypoxia, 4. duration of the procedure, 5. blood pressure, 6. bleeding, 7. infection, and 8. arrhythmias.

1. Anxiety

A certain degree of normal nervousness and concern about any dental procedure are bound to be present in most patients, if only because the patient doesn't know what to expect and is afraid of pain. Much can be done to keep this normal reaction from becoming uncontrolled anxiety, by proper explanation beforehand, in a brief, non-technical fashion, of what is going to be done, about how long it is going to take (overestimate is better than underestimate), what kind of discomfort the patient may expect, and what cooperation is required of him. A few minutes briefly spent in this fashion—even with a non-cardiac patient—would surely be repaid in most instances by a calmer, more cooperative patient. In the cardiac patient, one may help achieve some slowing of the pulse (or at least decrease the expected tachycardia), diminish the degree of sympathetic overstimulation of the heart, and prevent that degree of hypertension which has its origin in psychic stimuli.

In addition to conversation, one may make use of sedative drugs, but to rely on them solely is to use only half of your armamentarium. There is no point in my discussing the various available drugs. I am neither an anesthesiologist nor pharmacologist. Suffice it to say that one of the barbiturates, usually one of the rapid short-acting oral preparations, such as Seconal or Nembutal, makes an excellent premedication in association with a proper explanation of what the patient may expect.

2. Pain

In contrast to anxiety it is important to remember that mere conversation will not relieve pain. I would admit that anxiety may increase pain, but nothing relieves pain like adequate anesthesia. Again, I do not feel qualified to discuss anesthesia from the point of view of an anesthesiologist, but from a cardiac standpoint no anesthetic is contraindicated which permits an adequate concentration of oxygen in the inspired air. Certainly many dental procedures can be done adequately with procaine, and where the judgment of the dentist, not the physician, dictates the use of a local anesthetic, it is the agent of choice in a cardiac patient. If the dentist's judgment dictates otherwise, he should proceed with a general anesthetic. Inadequate control of pain leads to tachycardia, hypertension and increased myocardial irritability with a greater likelihood of the appearance of an arrhythmia; it may also cause muscular

struggling, particularly if barbiturates have been given preoperatively, and this in turn increases cardiac output and thus throws a greater load on the heart. This extra load may be enough to cause an attack of coronary insufficiency in a patient with arteriosclerotic heart disease, or of pulmonary edema in a patient with mitral stenosis. It is for this reason that no statement by a physician to the effect that a general anesthetic should not be used, should overrule the opinion of the dentist, who knows what agent will produce adequate anesthesia during the contemplated surgical intervention. Less urgent, but no less important—and for the same reason —is the postoperative control of pain by proper use of analgesic preparations.

In connection with the use of most local anesthetic preparations, there has been incorporated a vasoconstricting agent to help prevent too rapid absorption of the anesthetic. This agent is often epinephrine, and epinephrine is a drug which increases myocardial irritability, speeds the heart rate, increases over-all body metabolism and augments the force of cardiac contraction. For all these reasons, except perhaps the last, it is an undesirable drug in cardiac patients. In general, it is safer to give more local anesthetic, a more concentrated solution, or more frequent injections, rather than to use epinephrine. This is especially true in arteriosclerotic heart disease. Perhaps in young patients —under 40—with other forms of heart disease, the use of a dilute epinephrine solution (1:100,000) may be justifiable, but certainly not in cases of coronary arteriosclerosis.

3. Hypoxia

This is part of the total picture of general anesthesia. A cardiac patient—particularly class II or III cases, particularly arteriosclerotic heart disease in any class, particularly cyanotic or potentially cyanotic congenital cases—should always have 20 percent oxygen or more in the inspired air. This precludes the use of nitrous oxide in such patients, since adequate anesthesia cannot be obtained with this percent oxygen. Hypoxia causes increased myocardial irritability, tachycardia and hypertension, in addition to the direct effect of oxygen lack on brain cells and heart muscle. It is to be avoided at all cost. For most purposes, intravenous barbiturate anesthesia is satisfactory from a cardiac standpoint, as long as oxygen is available, and a bag apparatus to permit artificial respiration in the event of apnea. Careful premedication will do much to permit adequate induction without laryngospasm, but the dentist should be prepared to establish an adequate airway either trans-orally or by tracheotomy if necessary. The maintenance of such an airway is an important part of any general anesthetic, even in the absence of laryngospasm. Of the inhalation anesthetics, ether is the safest, but least convenient from the standpoint of administration. Cyclopropane is believed by some (but not all) to predispose to cardiac arrhythmias, particularly in the presence of epinephrine in the blood stream. From both the dental and cardiologic viewpoint, I think that an intravenous barbiturate in the hands of an experienced user is the most satisfactory and most convenient general anesthetic.

4. Duration

Also connected with the problem of anesthesia is the duration of the operation. Certainly it would be foolish to lay down any hard and fast rules in this regard. It would be unwise to subject a cardiac patient to many short operations, if two operations of an hour each would suffice. The extra risk associated with many inductions of anesthesia more than outweighs any benefit derived from a short-term load imposed on the heart. Likewise, a prolonged period in the chair, particularly when local anesthesia is being used, may be emotionally exhausting and would impose less of a load if divided into two or three shorter sessions. Here, as in so many places in medicine and dentistry, good clinical judgment — a thoughtful matching of the patient, his disease, and the solution to his problem—will suffice to determine the length of the dental procedure.

5. Blood Pressure

Many factors already discussed have a bear-

ing on this—pain, anxiety, hypoxia and anesthesia. The first three tend to raise the pressure, the last to lower it. Blood loss also is a hypotensive factor. Adequate control of all these factors will result in maintenance of a satisfactory pressure. The older sympathetic amines in common use for control of hypotension are contraindicated in cardiac patients, largely because of their effect of increasing myocardial irritability. If necessary, in the hypotension seen with general anesthesia, an intravenous drip of Noradrenalin, the non-methylated isomer of adrenalin, should be used. It has no irritable effect on the myocardium of the kind seen with ephedrine, neo-synephrine, or epinephrine; it is a good over-all vasoconstrictor unlike epinephrine; and it does not increase the heart rate. By regulating the rate of drip of a 500 cc solution containing 2 or 4 or 8 mg of Norepinephrine, one can easily maintain normotensive levels (or slightly hypertensive ones in a previously hypertensive patient). This is not indicated when hypotension is due to blood loss—but rather where it is due to a temporary loss of vasomotor tone. Obviously, with blood loss, blood replacement—or use of a temporary substitute such as plasma or dextran—is necessary. Prolonged hypotension is almost never seen with local anesthesia. What hypotension may occur is usually due to simple fainting and this could happen in no better place than the dentist's chair, where the chair can be tipped back and the head made slightly lower than the legs, until syncope is over. Probably further work on such a patient should be deferred until a later date, and the use of a general anesthetic considered.

6. Bleeding

I do not propose at this point to discuss the hematologic aspects of dentistry, but rather merely to talk of the management of the cardiac patient who has been receiving Dicumarol or Tromexan or one of the other cumarin preparations. There should be no surgery performed until the prothrombin time is normal and this can easily be tested. It usually becomes normal within 72 hours after the last dose of Dicumarol, and with 24 to 36 hours after the last dose of Tromexan. If emergency surgery is necessary, one may make use of Mephyton, 50 mg. by intravenous drip, which will produce a normal prothrombin time within 4 to 8 hours, provided that the last dose of anticoagulant has been given 24 to 48 hours previously. The other vitamin K preparations do not produce a normal prothrombin time until about 24 hours after they have been injected. Obviously, the best solution to this problem is to delay the surgery for 72 hours.

7. Infection

One could briefly dismiss the problem of the prevention of subacute bacterial endocarditis in patients with rheumatic and congenital heart disease with one word—Penicillin. Rather than be brief—something a speaker assigned an hour for a talk can never do—I shall add a few comments. Probably the easiest way to manage this problem is to arrange for a single injection—an hour before surgery—of a preparation which contains both the crystalline, short-acting Penicillin in a dose of 100,000 units, and a long-acting suspension such as Procaine Penicillin, in a dose of 300,000 units. All sorts of other dosage schedules are possible, including a long-acting drug the day before, the day of, and the day after surgery, or even frequent oral use in the 12 hour period on either side of the operative procedure. Almost any technique is satisfactory which provides an adequate serum level for 24 hours beginning immediately after bleeding has occurred. There is no value to any of the other antibiotics in this prophylactic procedure.

8. Arrhythmias

I have purposely not discussed the treatment of various arrhythmias. Their prevention is in the hands of the dentist, and proper avoidance of pain, epinephrine, hypoxia, and anxiety will prevent the occurrence of many. Only one other suggestion in this regard seems in order—i.e. not to use atropine preoperatively in patients with a history of paroxysmal rapid heart action. In the treatment of the specific arrhythmias, a physician should be called. The only ones which

are true acute emergencies are cardiac standstill and ventricular fibrillation. The hospital patient will be in an operating room with an anesthetist and should be within reach of a surgeon who can do the necessary thoracotomy, defibrillation if necessary, and then cardiac massage. The hospital should be ready for such an emergency and in rare instances, it may even be necessary for the dentist to do the deed. However, it would be most impractical for every dentist to set up his office for the handling of such an infrequent emergency. In that rare instance, I fear one would have to accept defeat.

This brings me to the end of the dental aspects of cardiovascular disease.

SUMMARY

Every dental procedure, just as every medical or surgical procedure carries some definite risk, however small, to the well-being of the patient. The object of a good dentist will be to keep this risk as small as possible. Certain forms of heart disease, particularly in certain stages, increase this risk, and it is this aspect of cardiovascular disease which is of interest to the dentist.

The problems facing a dentist consist in finding out if the patient has any organic disease, then recognizing the particular aspects which are of concern to him, and then knowing what to do about these aspects. The dentist cannot be expected to decide on the presence or absence of heart disease or to diagnose its nature. He can however, with a few simple questions, ascertain if the patient knows that he has heart disease, and often what kind. Except in most unusual circumstances, it should then be possible for him to obtain information from a physician as to the patient's cardiac diagnosis and the extent of the disability. This can be done by calling the patient's physician if he has seen him in the previous six months, or by asking him to visit his physician if this is not the case.

All forms of heart disease may result in the occurrence of an arrhythmia or congestive failure. The dentist should know what factors, that he can control, are likely to cause or exacerbate these problems. Coronary artery disease, in addition to these problems, includes the added risk of precipitation of angina or a coronary thrombosis by certain actions or agents employed by the dentist. Rheumatic and congenital heart disease are both associated with the special problem of subacute bacterial endocarditis, a condition about which the dentist also has it in his power to do something. In certain cases of either rheumatic or arteriosclerotic heart disease, long-term use is made of the anticoagulants. It is incumbent upon the dentist to know if such a patient is in his hands.

Having determined the existence of cardiac disease and its nature, having recognized the special facets which are of interest to him, the oral physician must know how to employ anesthetic and analgesic and antibiotic agents, so as to minimize anxiety, pain, hypoxia, the duration of the procedure, blood pressure changes, bleeding, infection and arrhythmias. In short, he must know how to perform his dental work so as to prevent or avoid the problems placed before him by the patient with heart disease.

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MORBO CARDIOVASCULAR E DENTISTERIA

Dr. med. Clarence Denton

SUMMARIO IN INTERLINGUA

Cata intervention dental—exactemente como cata intervention medical o chirurgic—implica un risco plus o minus leve pro le ben-esser del patiente. Le bon dentista cerca a reducer iste risco al plus basse grado possibile. Nonobstante, certe formas de morbo cardiac—specialmente in certe phases de lor curso—augmenta le risco, e iste aspecto del morbos cardiovascular es de interesse special pro le dentista.

Le problema con que le dentista se trova confrontate es (1) determinar si le patiente suffre de un morbo organic, (2) recognoscer le aspectos special que es importante ab le puncto de vista dental, e (3) saper lo que debe esser facite in re ille aspectos. On non pote requirer que le dentista sia capace a decider si o non le patiente suffre de un morbo cardiac o a diagnosticar su natura. Sed il es possibile pro le dentista poner alicun simple questiones pro determinar si o non le patiente mesme es conscie de suffrer de un morbo cardiac e in caso de si, de qual typo. Con le exception de circumstantias inusualissime, le dentista ha tunc le possibilitate de mitter se in contacto con le medico del patiente e informar se in re le diagnose cardiac del patiente e le grado de su incapacitate. Si le patiente ha essite sin consilio medical intra le passate sex menses, on deberea urger que ille re-consulta su medico.

Omne formas de cardiopathia pote resultar in arrhythmias o insufficientias congestive del corde. Le dentista deberea saper per qual medios ille pote controlar le factores que es le plus probabile causas del precipitation o exacerbation de iste problemas. In casos de morbo del arteria coronari il ha—ultra le complicationes mentionate—etiam le risco que le activitate del dentista o le agentes chimic empleate per ille pote precipitar un angina o thrombosis coronari. Le cardiopathias rheumatic e congenite es associate con le problema special de subacute endocarditis bacterial, sed isto es etiam un problema relative al qual le dentista non es disproviste de potentialitates. Post determinar le presentia de un cardiopathia e, in plus, post recognoscer le aspectos special que es de interesse odontologic, le medico oral debe saper como usar agentes anesthetic, analgesic, e antibiotic e como executar su labores dental sin causar o precipitar ulle complicationes in le stato del patiente cardiopathic.

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BRUXISM: A REVIEW AND A CASE REPORT

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Bruxism is usually defined as the grinding of teeth during sleep. The term is reported to have been used first by Samuel C. Miller, having been derived from the word bruxomania. The latter term, according to Isador Hirschfeld, is derived from la Bruxomanie, and was first used by Marie and Pietkiewix in 1907 (Boyens). Other terms for the same condition are kardi effect, stridor dentium, and occlusal neurosis. Bruxomania is definied as the grinding of the teeth as a manifestation of neurosis, usually occurring during sleep. Most authors do not make any distinction between the two terms, bruxism and bruxomania, as they are usually grouped together in discussion. In fact, Boyens' definition includes inaudible clenching, audible gnashing, cusp tripping and abnormal contacts made during the most extreme and unsuspected mandibular excursions. Occlusal neurosis, a term relevant to this discussion, was defined by Tishler as the grinding, pounding, or setting of the teeth, when the mouth is empty. Leof refers to such conditions as clamping and grinding habits.

ETIOLOGY

The cause of bruxism and related conditions can for the most part be classified under three main headings: (I) psychic factors, (II) intraoral mechanical factors (occlusal imbalance), and (III) extraoral organic factors.

PSYCHIC FACTORS

The psychic factors are the most important and are almost always present, despite the fact that the intraoral mechanical factors usually receive all the emphasis. The psychic factors which cause or contribute to bruxism generally include emotional tension in the form of unrealized aggression, neuroses, and deleterious habit patterns. As one can readily see, bruxism should, in most cases, be classified as a psychosomatic illness.

"The theory that there is a relationship between the psyche and the soma, the mind and the body, is not a new one. As early as 380 B.C. it was expressed by Aristotle, who wrote, 'Probably all the affections of the soul are associated with the body; anger, gentleness, fear, pity, sorrow, and joy, as well as loving and hating, for when they appear the body is also affected.' However, it remained for Freud to supply the stimulus which evolved into the modern concept of psychosomatic medicine, and, from the point of view of scientific thought, the beginning of the psychodynamic approach must be credited to him and his theories that a great part of human behavior is determined by unconscious motivation." (Ash)

The term psychosomatic is derived from the original psysomatic coined by George Draper some thirty years ago (Miller). The American Illustrated Medical Dictionary, twentieth edition, defines *psychosomatic* as "bodily symptoms of a psychic, emotional or mental origin." Psychosomatic medicine is a relatively new field. A tremendous impetus was received from World War II due to thousands of emotionally induced physical disturbances (Grinker and Spiegel).

Some of the illnesses now known to be either caused by or greatly influenced by psychosomatic mechanisms are peptic ulcers, gastritis, duodenitis, ulcerative colitis, hypertension, recurrent neurodermatitis, urticaria, bronchial asthma, headaches, nail-biting, and hyperthyroidism. Just as psychosomatic medicine is being given its just recognition, the field of psychosomatic dentistry is also assuming its rightful position.

Miller defines psychosomatic dentistry as the relationship of the mental well being to the

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health and integrity of the oral tissues. Stolzenberg quotes "It is by no mere coincidence that the dentist is faced by such problems as gagging, retching, fear of dental service, burning sensations of the tongue, excessive salivation, alterations in the salivary pH, teeth gnashing and bruxism. Nervous tensions and psychogenic repressed factors contribute tremendously to the psychosomatic problems in dentistry."

Emotional Tension: Campbell, in an excellent dissertation on psychosomatic mechanisms, quoted Sherrington, a great English neurophysiologist, as saying, "Emotion moves us, hence the name." Campbell further states that "moved to tears, paralyzed by fear, tense with expectancy, breathless with excitement, gnash our teeth with rage, raging headache, burning tongue, a foul mouth, sick with fear, griped by someone's gall, impotent with rage, are all expressions of emotion." He states that "emotional reactions have their origin in quite primitive biologic mechanisms concerned with food getting, with protection from danger, and with reproduction of the species; in other words, they occur in those organismal reactions most clearly related to survival of the individual and of the race—hunger and sex. In humans they become very complex, because in addition to being directly aroused by external threats to life (such as starvation, conquest by enemies, or deprivation of affection) they may be activated indirectly by symbols or cues which have come to stand for, or represent, such external situations. Subjective states of fear or anger, together with their somatic expressions in the form of trembling, dilated pupils, tumultous heart action, clenched teeth, dry mouth, and other visceral reactions, may be produced in response to a real environmental danger or to the thought or memory or symbol of one. Memories, phantasies, the anticipation or misinterpretation of facts can all arouse as strong (and sometimes stronger) emotional reactions as can actual happenings in the external world. Anxiety is produced then, when our impulses clash with the external realities of social existence or when they conflict with each other. Drives for sexual gratification not only clash with morality, but their aggressive aspects are incompatible with coexisting tender, protective, altruistic trends within the personality. The tension thus produced can lead to such alarming symptoms as frigidity, impotence, and other troubles. Continual stimulation of the emergency system of the body can produce high blood pressure, ateriosclerosis and, eventually, heart disease."

Unrealized Aggression: Rittenburg, as quoted by Miller, explains the etiology of bruxism by saying "Within the personality there exists an aggressive group of instincts. They strive to find expression in the outside world. They would destroy, they would engulf, they would annihilate the outside world if they could get there. We can occasionally see this happening in pure form, in the manic rages of the insane. But under ordinary circumstances, the world of personal and social reality sets up barriers to the unchecked gratification of these instincts, in the form of conscience, morality, education and training. When the dynamic relationship of aggressive drives and repressing forces begins to show strain, the muscles of the mouth may begin to show irrational phenomena. They may contract unconsciously in response to the 'leaking out' of aggressive instincts, and bruxism results. In every case of grinding of the teeth, there is a defect in the measures which the personality employs to inhibit aggression. In every case of bruxism the core of the problem is to be found in the functioning of the personality. The somatic destruction is the avenue of expression for the psychopathology which determines the why of the disease."

Ewalt gives some insight as to the incidence of these conditions by stating, "Every patient has somatic and psychological problems, excepting those that are dead when the doctor arrives."

Neuroses: Blakiston's New Gould Medical Dictionary, First Edition, defines anxiety neurosis as a "psychoneurosis characterized by emotional instability, irritability, apprehension, and a sense of utter fatigue; caused by incomplete repression of emotional problems. It is associated with visceral phenomena such as tachycardia, palpitation, nausea, a sense of suffocation, diarrhea, tremors, and perspiration." Borland says "The term 'neurotic' has many un-

warranted fearsome connotations for many people. We all tend to resent its application to our own behavior and many of us are very reluctant to apply it to the behavior of others. The word really refers simply to the tendency to display, in certain situations, habitual behavior patterns or feelings which are less satisfying than other more appropriate reactions. Interpreted in this sense, the word is applicable at times to all of us."

Massler and Malone, in a review of nailbiting, said "All children are subjected to a bombardment of 'musts' and 'must nots' and live in an environment of frequent frustrations which inevitably create tensions. When those frustrations are mild and the child is an adaptable one, a 'normal' situation results. If the tensions are severe and the child is not very adaptable, neurotic tendencies begin to appear."

Habits: Sorrin, in an outstanding article on habits and their relation to periodontal disease, said that "habits, in general are actions which at first were deliberate and occasional, but which throughout repetition became subconscious and reactive." He contributes a very excellent classification of habits. He groups any abnormal occlusal habit, or occlusal and incisal grinding, and clenching of teeth in control of emotions, under habit neuroses. Under occupational habits he incorporates any occupation in which, through concentration on work, the patient is found grinding the teeth in rhythm with the work at hand. Leof has also listed various clamping and grinding habits but Sorrin's outline is the most complete.

In their text on periodontia, Stillman and McCall state that "habits of occlusion are usually the results of nervousness and may cause injury in the case of teeth whose occlusion in the normal movements of the jaw is innocuous. This is due to the fact that the patient tends to place the mandible in an unusual position in order to repeat an occlusal contact first occurring by accident only, thereby establishing a new occlusal habit."

INTRAORAL MECHANICAL FACTORS

Tishler discussed mechanical and psychic factors together because he recognized that the two are inseparable. Just as periodontoclasia is influenced by both local and systemic factors, bruxism can be modified or precipitated by psychic and/or mechanical factors. In other words, mechanical factors play an important role in the etiology of bruxism, but without the proper emotional background bruxism will not result. Likewise, a patient can very easily possess the proper emotional predisposition for bruxism, but may lack the mechanical catalyst, such as a premature contact, which could attract attention and initiate a grinding neurosis on either a conscious or unconscious level. Tishler lists the following mechanical conditions which contribute to bruxism. (1) occlusal trauma, (2) extra-alveolar leverage, (3) hypo-occlusion, and (4) tooth morphology.

Occlusal Trauma: "Even mild cases of occlusal trauma or minor anatomic defects as sharp cusps, will receive undue attention by the neurotic individual, resulting in bruxism." He also stated that a large precent of patients whose normal psychic impulses are easily unbalanced often develop the habit of continually grinding or abnormally pressing the teeth together with much force. This is undoubtedly induced by a feeling of discomfort due to traumatic occlusion. The habit neurosis is most frequently practiced during sleep, periods of worry, excitement, unusual stress or effort. It is found that such patients are inclined to magnify the importance of ordinary events, which keeps them in a state of constant mental unrest and likewise exerts an influence on the physical fatigue of which they often complain. Tishler says, "The habit of grinding or pressing the teeth together occurs so frequently in connection with such cases that the writer believes traumatic occlusion to be a contributing factor of major etiological importance."

Extra-Alveolar Leverage: One of the conditions responsible for the development of an occlusal habit neurosis is that which Gottlieb terms an extra-alveolar leverage. He believes that the teeth constantly move occlusalward during life and that some erupt more rapidly than others. This circumstance results in traumatic occlusion.

Hypo-Occlusion (Open Bite Case): It has

been observed that bruxism occurs is some patients who have an absence of centric occlusal contact in the incisal region. In an unconscious effort to establish contact in this region, they may grind their teeth until contact is established.

Tooth Morphology: There is a morphological factor involved in the practice of habit neuroses. This may be illustrated by considering those bicuspids and molars with long pointed cusps developed at an acute angle to one another with an intervening, narrow intercuspal space. Because of the existing occlusal relationship, the buccolingual and linguo-buccal mandibular movements are markedly restricted and confined entirely to a limited area on the cuspal surfaces. In other words, the occlusal movements are more or less "locked" and the teeth thereby deprived of the free, easy motion which normally permits cusp points to slide past one another and distribute occlusal stresses evenly.

Traumatic Occlusion, Malocclusion, Dentigerous Cysts: In speaking of bruxism in children, Miller lists these factors. He says "that although children often grind their teeth during sleep because of stomach disorders, nervousness, or psychogenic disturbances, traumatic occlusion, malocclusion, or dentigerous cysts may be responsible for this condition." More precisely, it is the combination of the local irritating factors and the emotional factors which produce bruxism.

EXTRAORAL ORGANIC FACTORS

This aspect of the etiology of bruxism, like the preceding two divisions, is also not clear-cut. Actually, there is an inseparable relationship among all these factors.

Endocrine Dysfunction: Wolf says, "An overactive thyroid gland may cause great nervous tension which in turn may cause bruxism." Miller states, "An abnormal habit may be produced by endocrine dysfunction, and in these instances a careful examination of the patient is advised. For example, an overactive thyroid gland may result in great nervous tension which in turn may lead to bruxism."

It has been stated that psychic factors are very important in the etiology of thyroid disorders, especially hyperthyroidism (Ewalt, Means). Here we have a vicious cycle where the psychic factors set the stage for bruxism. The increased irritability produced by the bruxism in turn aggravates the hyperthyroid state. Thus a patient who already has difficulties that he cannot handle is faced with another problem; that is, bruxism, or loss of teeth due to bruxism. This, of course, augments the nervous tension, which in turn increases the severity of the thyroid condition.

Nervous Reflexes: Miller states that bruxism may be due to nervous reflexes. For instance, a person with a chronic gastrointestinal disorder may be so stimulated as to cause him to grind his teeth. Along this same line of thought, Miller states that "bruxism in children is considered to be due to vesical irritability, hyperacidity of urine, or rectal parasites." Obviously, any of these conditions can initiate a nervous reflex over a period of time. Matthews says it is common to find bruxism in nervous children who are suffering with helminthiasis.

SIGNS AND SYMPTOMS

Bruxism is associated with periodontal disease in most cases. Bruxism should be suspected when (1) teeth are worn more than is normal for the age of the patient, (2) when the chief complaint is sore teeth or aching jaws upon awakening, (3) when the restorations are unaccountably broken, (4) in all cases of alveolar bone resorption (Ingersoll). Another sign is the presence of an incisal or in some cases cemental sensitivity to hot or cold or acid substances. The incisal sensitivity will, of course, be due to failure of the pulp to lay down secondary dentin as fast as the incisal dentin is being worn off. Sensitivity of cementum may be due to a referred pain from the periodontal fibers which have been traumatized and irritated because of the abnormal stresses of the grinding or clamping habits. Boyens reports that sore teeth upon awakening and gingival bleeding at night are frequent signs of bruxism.

Leof says that the existence of even some of the following symptoms at the moment of

awakening is usually proof that the patient has been clamping or grinding. These include a throbbing soreness of the teeth which disappear a few minutes after awaking, a consciousness of teeth in any way, teeth in contact, sensation of looseness, a feeling of tension in the region of the antra, strained muscles of mastication, or a consciousness of the temporomandibular joint.

Glickman says that bruxism may be attended by temporomandibular joint disturbances as the result of trauma from continual grinding or excessive overclosure due to abnormal wear, inadequately compensated for by continuous eruption. The disturbances which may result from such occlusal disharmonies vary in intensity depending upon the severity and duration of the trauma and the degree of compensatory adaptation which occurs in the joint.

DIAGNOSIS

"Unfortunately, the diagnoses of bruxism are as varied as the origin. These conditions are being diagnosed as Vincent's infection, Vincent's angina, trench mouth, gingivitis — all of the common disorders — and are being treated on the supposition that destruction of the organisms will clear up the disorder. We have demonstrated, as have many others, that this theory of medicinal application and oral hygiene is unsuccessful." (Ingersoll)

The diagnosis of bruxism is not an easy one. A large number of patients so afflicted are completely unaware of it. Leof helps establish his diagnosis in the following manner. He asks direct questions on the first visit. "Do you clamp your teeth? Do you grind your teeth?" "In a small percentage of cases, the answer is in the affirmative. Usually, however, the patient is puzzled and cannot answer or emphatically denies the possibility of any such habits. This response should not deter the practitioner. A brief explanation is offered the patient. The patient is told that many persons have habits of which they are unconscious, and that the unconscious existence of a habit is no indication of wrongdoing. The explanation that such habits are common and can be eliminated by conscious effort often aids the patient in overcoming any mental resistance which might hinder an attempt to disclose the habits. It also tends to create in the patient a realization of the damage that he may be causing his teeth through unknown habits. It will arouse his curiosity and interest and thereby lead to his cooperation. . . . By assuming the role of a vigilant and impartial observer, the patient may be able to clearly establish in his conscious mind all the habits in which he may be indulging unconsciously. After one week of observation, the patient is usually conscious of daytime habits. His difficulty comes with an attempt to recognize and describe night habits. . . . Where there are doubts, he is asked to check for the symptoms of bruxism at the moment of awaking. Often in nocturnal grinding, the noise of the gritting and sliding of the teeth is quite audible, so that someone sleeping near the patient can easily discover the habit. Where the patient may be a "clamper", we ask that he have someone gently part his lips while he is asleep and note the position of his teeth."

Boyens employs essentially the same procedure except that he has the patient repeat to himself before going to sleep, "I will awaken if I bite or clench my teeth." Several months may be required for some patients to accomplish this. He has found that the waking suggestions often "carry over" and are thus also effective during sleep.

Some patients complain of becoming "tooth conscious", but that is the lesser of a number of evils, and usually they recover from it in a comparatively short time (Boyens-Leof).

It appears then, that to be able to make an accurate diagnosis of bruxism, one must have a knowledge and appreciation for psychosomatics, and be able to obtain an adequate history from patients who may be unaware of the habit.

PHYSIOPATHOLOGY

In order to understand the effects of bruxism, it is necessary to have a knowledge of the physiology of the normal periodontal membrane and related tissues. The following discussion was taken from Leof's paper on clamping and grinding habits.

NORMAL FUNCTION OF PERIODONTAL MEMBRANE

The periodontal membrane functions as a cushion between the inelastic tooth and the comparatively inelastic alveolar bone. It supports the tooth in its position against movement through the pull of its fibers in one area, combined with resistance to compression of the membrane in an opposing area. When we chew food and bite on the teeth with 40 to 150 pounds pressure per square inch, the food buffers the shock slightly, but when the force reaches the tooth, it is the periodontal membrane alone that cushions the shock, i.e., gives under the force. In normal chewing, there is a combination of the lateral motions and the vertical or up and down motions of the jaw. This vertical motion is responsible for the intermittent quality of the force upon the tooth. In this momentary contact and release of normal function, the fibers of the periodontal membrane are intermittently stretched and released and the thickness of the periodontal membrane increases and decreases as the tooth moves in function. This normal function is conducive to the health of the periodontal membrane.

ABNORMAL FUNCTION IN BRUXISM

Destruction: In the habit of clamping and grinding, which manifests itself with numerous variations, the force applied to the tooth is almost always sustained rather than intermittent. Under the sustained pressure, groups of periodontal fibers are under constant tension without release or rest. In opposing areas, the periodontal membrane is compressed for periods of time without release. This prolonged tension and compression may cause congestion and irritation of the periodontal membrane as differentiated from the stimulating effect of the intermittent force. The repeated forces of habit origin can, in time, seriously damage the periodontal membrane, causing periodontal disease, or they may become a secondary factor in an already existing periodontal disease.

Orthodontic researches (Oppenheim) have proved that light sustained force, i.e., in ounces, will cause tooth movement without development of pathosis in the periodontal membrane. However, when the sustained pressure of an orthodontic appliance is increased beyond a critical amount, there is degeneration of the periodontal membrane or resorption of the root and possibly destruction of the alveolar bone. Yet the amount of sustained pressure which these studies have shown to be dangerous to the periodontal membrane is only a small fraction of the pounds per square inch applied in grinding habits. The slight amount of this pressure leads to the conclusion that it is the sustained quality of the pressure in the clamping or grinding habits that is its most harmful characteristic.

Three properties of the force relayed to the periodontal membrane in clamping or grinding habits differentiate it from normal function:

1. The constancy of its force (the fact that it is not intermittent.
2. The excess force in pounds
   (a) The total muscular force may be applied on a small group of teeth because of extractions or the particular character and direction of the habit and
   (b) The muscle strength of the individual may be overdeveloped as a result of the clamping and grinding habit.
3. The excessive time per day that the force is applied.

Coolidge says that "where the periodontal tissue is continuously traumatized but not entirely destroyed, degeneration occurs in the form of hyalinization of this tissue. In a histologic study of the periodontal tissues of fifteen human jaws, he found that abnormal or excessive stress resulted in pathologic changes similar to those seen in animals where excessive orthodontic forces were applied. These changes were hemorrhage, thrombosis, hyaline degeneration and necrosis of the periodontal tissue, excessive bone resorption, root resorption and fractures of cementum from the root surfaces.

In most cases bruxism causes and perpetu-

ates a destructive process. Rosenburg says that "bruxism may eventually lead to a vicious cycle by causing paradontosis, thus impairing mastication and causing malnutrition. The malnutrition in turn leads to failure of other organs and a general malaise, causing increased feeling of concern and inferiority, and further emotional unbalance, potentiating the bruxism."

Stimulation: However, in some few cases, there may be a stimulation of growth and development of the periodontal tissues due to bruxism. Miller says the amount and type of tooth function determines the structures of the alveolar and supporting bone. Leof has reported that in some cases of excessive grinding (bruxism), the hyperfunction may even result in a periodontal condition of greater health than average, i.e., greater density of alveolar bone and better health of gingival margins. This does not occur with clamping habits.

INCIDENCE

There have been no studies of statistical value concerning the incidence of bruxism. There has been, however, considerable clinical evidence reported which indicates a definite relationship between bruxism and periodontal disease. Boyens reports that almost 100 percent of periodontal patients are or have been bruxists in some form. In a study of 100 of his periodontal patients, 78 were found to exhibit bruxism.

Leof agrees by stating that "clinical observation has demonstrated that the majority of periodontal cases have, as a significant factor, some pressure habit or habits, either as an initial or as a contributory cause." He made a thorough study of 171 periodontal cases with habit involvements and followed 52 cases for five to ten years after treatment. He found that clamping habits were the most common, with grinding habits next and finger nail biting, wrist biting, etc., the third most common. The incidence of clicking the teeth together or chewing on foreign objects such as pencils or spectacles was the least significant habit in these cases.

Matthews, interested in the incidence and treatment of bruxism among school children, reports that "enquiry from school medical officers reveals that the condition is well known, but no active steps are taken to correct it. Further enquiry reveals that it is not uncommon among adults, judging from comments of army doctors."

TREATMENT (MECHANICAL)

Restoring Lost Vertical Dimension: As it was previously mentioned, most of the treatment for bruxism has been of a mechanical nature. This treatment usually is an attempt to restore the vertical dimension by various mechanical means. As one might suspect, the habits usually persist and the patient either chews through the restorations or develops periodontal disease due to the abnormal stresses.

Occlusal Equilibration: More success has been noted in cases where a traumatic occlusion is the precipitating cause. Tishler said that the habit of grinding or pressing the teeth together occurs so frequently in connection with traumatic occlusion that he believes it to be a contributing factor of major etiological importance. He adds that the proof of this contention lies in the fact that an occlusal adjustment, which distributes the occlusal forces more equitably, results in an immediate disappearance of the habit. Sorrin, Miller, Susman and many others agree that occlusal equilibration should be an integral part of the treatment for bruxism. Even if this does not stop the habit, it will at least distribute the stresses more evenly, helping the prognosis somewhat.

However, Boyens says that it is impossible to remove all potential causes and even then the habits may be of such long duration as to have become an unconscious habit.

Construction of Splints: The next most common mechanical treatment is that of splint construction, usually made so as to immobilize the mandible during sleep. Although some authorities indicate that this method is used routinely (Susman, Ingersoll and Kerens, Kaner, Glickman and Matthews), it is employed with caution as abnormal stresses can still be brought to bear on the teeth and periodontal tissues. For example, Leof says that

immobilizing splints are to be used only as a last resort and then only for not longer than two months to break the habit. Matthews has published a technic for the construction of rubber latex splints because he believes the acrylic splints are deleterious to the tissues. Kaner offers a technic for construction of a combination acrylic-vulcanite splint. Ingersoll and Kerens propose a technic for construction of a semi-soft vinyl resin splint. It is said to be not so hard as to cause damage nor soft enough to allow the patient to still practice the grinding habits.

These splints may serve several functions. First, they may simply prevent any movements from taking place but this is not likely. Secondly, and probably more important, the presence of the splint in the mouth and its resistance when movement is attempted probably serves as a reminder to the patient that he should not attempt to grind his teeth. Thirdly, the points of interference or trauma due to faulty occlusion cannot be reached and concentrated on, thereby the patient receives no enjoyment nor satisfaction from his grinding.

PSYCHOTHERAPY

History Taking: "If the history serves no other purpose, it makes the patient feel that the dentist is interested in him as a person and does not consider him as just a mouth full of teeth to be filled or extracted" (Chavoor). Campbell says that "the one essential tool for exploration of this field (psychosomatic oral disease) is good history taking. Not only is a circumscribed disease process studied, but the sequence of events in the person's life leading up to and including the time of onset, such as states of worry, fatigue, frustration, etc., in business and family relationships, should be sought and thoroughly investigated. It will be amazing how frequent is the association of prolonged financial worry or marital incompatibility with dental caries or paradentosis." Both Cobb and Dunbar provide excellent technics for psychosomatic history taking.

Actually, the history taking is the most important step in both diagnosis and treatment of occlusal habits. Without a proper history the diagnosis, and consequently, the treatment of the condition will be incorrect.

Autosuggestion: Boyens says that the conscious mind makes use of, and controls, the unconscious mind to a large extent. To exercise this control effectively for our therapeutic purposes, we can make use of autosuggestion. Autosuggestion involves the repetition of a fixed and positive thought, worded in such a manner that the unconscious reaction is one of harmony and in accord with conscious demands. His first suggestion is used to help establish the diagnosis in patients who are unaware of any occlusal habits; i.e., "I will catch myself if I bite or clench my teeth." The next suggestion, to be used to establish a diagnosis for night grinding is "I will awaken myself if I bite or clench my teeth." If the patient suffers with insomnia, he gives daytime suggestions only. This form of autosuggestion makes, or tends to make patients conscious of a habit which heretofore has been unconscious. After the patient is aware of the habit, he can stop it voluntarily. Boyens first tried negative suggestions such as "I will not bite my teeth" but had little success with these suggestions. While he used autosuggestion specifically to check bruxism, he adds that there is the probability that other unconscious habits, such as lip or nail biting and thumb sucking can also respond to this type of treatment.

Leof describes a treatment plan somewhat similar to Boyens'. The patient is made conscious of his habits and is told to substitute the normal rest position for the abnormal habits. For night grinding, a relaxation routine is set up and the patient is observed by a member of the family for results. If this does not work, a change in sleeping position is tried. Sleeping on the back without a pillow tends to pull the mouth open and gives good results. If this is not effective, an alarm clock is used to wake the patient every three hours to remind him to relax his jaws. All this tends to bring an unconscious habit to a conscious level where it can then be controlled.

Referral to Psychiatrist: Borland states that attempts on the part of the dentist to diagnose and treat before such resolution has occurred are almost certain to fail, but a cooperative approach by the dentist and his colleague, the

psychiatrist or psychologist, offers a good prognosis. Borland indicates that it is very easy in most situations to explain that problems as worry, tension, and emotional duress often make pain and other sensations more noticeable and less easily tolerated. With this approach, a psychological referral can be accepted by most patients without resentment or defensiveness.

CONCLUSIONS

Bruxism can, in most instances, be considered as a finding of a much greater and more burdensome underlying emotional disturbance. The relief given by removal of such conditions, with subsequent correction of occlusal habits, is inestimable.

"The application of psychosomatic principles to dental medicine is a field wide open for exploration. As long as physicians, surgeons, and dentists limit themselves to their special treatment of specific organs or systems, they will never see the patient either as a total organism of interrelated organs and systems, or as a member of a community. The 'oral cavity' is a verbal fiction as a moment's consideration shows." (Campbell)

Likewise, Burstone says, "It is apparent that there are certain psychogenic factors in the etiology of dental ills. Dentistry, if it is to fulfill its obligation as a health service, must be prepared to recognize the possibilities of a psychic as well as a physical and physiochemical etiology in dental disturbances. From the therapeutic viewpoint, this means that attempts to treat by purely mechanical means, conditions in which there are psychic components, are inadequate. We must, therefore, make every effort to equip ourselves with the knowledge necessary to understand the relationship between the psychic and somatic processes in dental health and disease."

CASE HISTORY

Mrs. R. O. McD., a forty year old housewife, was referred to the University of Alabama Dental Clinic April 8, 1953. A physician who had previously examined her recommended the extraction of her remaining teeth to alleviate her complaints of "backache, earache, and jointache."

Oral examination revealed a marked generalized attrition with an estimated 6-7 millimeter loss of vertical dimension. It appeared that probably one-third or more of the clinical crowns of the anterior teeth had been worn away. The enamel and dentin was normal in so far as physical characteristics were concerned. All first molars and the mandibular second molars were missing. The maxillary second molars were extruded. The remaining teeth exhibited very little evidence of caries experience. There was a considerable amount of calculus present but the periodontal condition was

Fig. 1. Profile view illustrating loss of vertical dimension, fissuring at the corner of the mouth, and thin lip line.

average or possibly better than average for a person this age. Full mouth roentgenograms indicated a relatively normal periodontal condition, with no caries and no apical pathology being observed. Areas of pulp calcification could be seen in all the teeth and were particularly noted in the lower anteriors.

The patient's facies was that of an edentulous person. The chin had assumed a protrusive position; the lips appeared as thin lines; and the corners of the mouth were deeply fissured. The patient's manner was rather strained. She looked uncomfortable and tired, and seemed to be somewhat reluctant to ask that her teeth be extracted.

According to the patient, her bruxism began about eight years ago, with the grinding habit being practiced only during sleep. For the past five years nocturnal bruxism has been so pronounced that her husband has not been able to sleep in the same room with her because of the noise. The patient also reported that her teeth have been very sensitive to thermal changes and to acid foods. For this reason she has refrained from eating certain foods such as ice cream and apples. The abnormal sensitivity was probably due to the inability of the pulp to form secondary dentin as fast as the tooth structure was being worn away.

The patient had noticed that upon awakening her jaws were "tightly closed, tired and stiff." Her teeth were "sore." Movement of her lower jaw caused "popping, clicking and grinding noises." Palpation and auscultation of the temporomandibular joints indicated that both joints were involved in producing the noises and discomfort. The patient reported that for the last eight years she had also been bothered with earaches which, in the last five years, had become more or less constant.

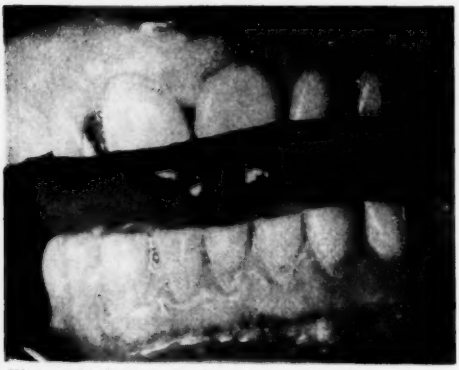

Fig. 2. Reduction in length of clinical crown, exposure of dentin.

Throughout the history taking, a possible cause for the grinding neurosis was sought. The patient insisted that she had no more complaints nor burdens than the average person. But at the same time she revealed that almost every afternoon she would become so depressed that she would cry for two or three hours for no reason at all. She also reported that she had a "nervous bladder", which required relief every half-hour or so during the night and day. Chronic depression and loss of sleep kept her in a state of exhaustion at all times. Her physician had treated her unsuccessfully with vitamins and sedatives.

The dental examiner* realized that the extraction of the remaining teeth and construction of dentures would not solve this patient's problems. In fact, the loss of her natural teeth would have, in all probability, depressed her even further. The patient indicated that she would be willing to undergo psychotherapy and was therefore referred to the Psychosomatic Clinic of the Jefferson-Hillman Hospital.

Fig. 3. Generalized abrasion of the maxillary teeth with pronounced dentinal cupping due to fast rate of wear.

The patient was seen in the Psychosomatic Clinic for a total of about four hours divided into thirty-minute appointments. The eight visits were spread progressively further apart. Psychotherapy, except for follow-up checks, was completed in about four months.

The attending psychiatrist† recorded the following notes in Mrs. McD.'s clinic chart. "At the first visit, Mrs. McD. was tense, apprehensive and considerably depressed. The Rorschach

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test confirmed the depression. She was sleeping fairly well, but fitfully waking up during the night to empty her bladder. Her husband had told her repeatedly that while she slept, she would grind her teeth to the extent that it annoyed him. The patient was not conscious of grinding her teeth during the daytime.

Further questions revealed that Mrs. McD., although middle aged, was still closely attached to her mother and the chief point of disturbance was her mother's control over Mrs. McD.'s children. The mother lived close by and would frequently come to visit. She had a rather dictatorial manner, and would give the children harsh corrective discipline which made Mrs. McD. resentful. Mrs. McD., having been reared

Fig. 4. Acquired centric showing loss of vertical dimension.

under the same strict discipline, dared not oppose her mother for fear of offending her. The fact that Mrs. McD. had a son in the Armed Forces did not seem to disturb her as much as her mother's attitude. Mrs. McD. said she could not stand the situation, but that there was nothing she could do about it.

The psychiatric examiner believed that Mrs. McD.'s depression was simply a reaction to this situation rather than being a true depression. Since the examiner felt that good rapport had been obtained during the first interview, amphetin compounds were prescribed to control the depressive element and mild sedation was prescribed to improve the sleep pattern. Psychotherapy in the form of insight and reassurance were utilized to help Mrs. McD. relieve the tense situation between herself and her mother. It was explained that her elderly mother perhaps still considered Mrs. McD. a child, and therefore assumed considerable responsibility for the rearing of Mrs. McD.'s children. It was further explained that the grandmother's behavior might be partially caused by certain degenerative cerebrovascular changes, which commonly occur in senile individuals.

Mrs. McD. was advised to discuss the point of irritation with her mother in a pleasant, ladylike, but firm manner. She was encouraged to do this unemotionally, as it was pointed out that one cannot be emotional and think clearly at the same time. The examiner recognized the fact that Mrs. McD. did not wish to hurt her mother by being rude, abusive or resentful.

Fig. 5. Maxillary study cast with acrylic splint in position.

However, it was pointed out that Mrs. McD.'s mother would have to understand that her interference was the chief cause of Mrs. McD.'s poor health.

The patient was seen at frequent intervals for a few times and began to improve as far as her depression and sleeping were concerned. Following the psychiatrist's advice, Mrs. McD. had openly discussed the problem with her mother, presumably in an unemotional, tactful, but firm manner. At first the mother was extremely offended, and this in turn further upset Mrs. McD. However, Mrs. McD.'s mother soon began to interfere less and less. The children particularly responded to the relief of their grandmother's disciplinary measures and became more happy. This pleased Mrs. McD.

and helped her maintain the position she had taken in regard to her mother. Eventually, both women became adjusted to a new relationship.

With continued reassurance and support Mrs. McD. continued to improve and seemed to enjoy coming to the clinic to talk with us. We could never discover that she had been unkind to her mother. Apparently she had followed well our instructions in handling the problem with her mother. As the patient improved and felt better, her whole personality changed. Mrs. McD. stated that as long as she thought she was doing the right thing, she did not worry about her mother's attitude. She finally recognized that her mother's behavior was that of an immature, elderly person.

It was thought that psychotherapy brought considerable comfort and relief to this patient. The problem involved a middle-aged woman who had never achieved a normal degree of independence from her domineering mother. This case seems to indicate that bruxism may have an emotional component and can be classed as a psychosomatic illness.

On the patient's third visit, an acrylic splint which covered the palate and occlusal surfaces of the maxillary teeth was given to her with instructions to wear the appliance at night. On each subsequent visit the splint was examined for signs of abrasion. The marks and scratches were then removed by polishing. The patient reported that the splint was not uncomfortable and that she "lost" it only two nights. For the first three or four weeks the splint showed considerable evidence of night grinding. However, this diminished until at the end of three months there was no evidence of any night grinding. In addition, the patient reported that her teeth were less sensitive and that for the first time in five years she could bite into an apple without suffering pain.

Although this case has been followed for only one year, it appears that Mrs. McD. is now a normal, happy person. She has been relieved of her "backache, earache, jointache." She sleeps soundly throughout the entire night, and she no longer experiences the afternoon periods of depression. Following completion of the psychotherapy, Mrs. McD. was happy to be referred to the dental clinic for full mouth rehabilitation rather than for full mouth extraction.

It is our opinion that the service rendered this patient indicates that a proper diagnosis must of necessity consider the psychosomatic aspects of dentistry.

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BRUXISMO

Dr. med. dent. Edward W. Strother e Dr. med. dent. George E. Mitchell

SUMMARIO IN INTERLINGUA

Bruxismo es normalmente definite como le strider del dentes durante le somno. Altere terminos pro le mesme condition es bruxomania, odontoprisis, stridor del dentes, e neurosis occlusional.

Le causas de bruxismo pote esser classificate in tres categorias principal: (I) factores psychic, (II) factores mechanic intra-oral (disequilibrio occlusional), e (III) factores organic extra-oral. Le factores psychic es le plus importante e se manifesta in quasi omne casos. In general illos include tensiones emotional como effecto de aggressivitate sin abreaction, neuroses, e nocive habitudes de conducta. Le factores mechanic intra-oral include (1) trauma occlusional, (2) impacto vectal extra-alveolar, (3) hypo-occlusion, e (4) morphologia dental. Le factores organic extra-oral es dysfunctionamento endocrin, specialmente hyperthyroidismo, e reflexos nervose.

Le signos e symptomas de bruxismo es (1) dentes anormalmente abradite, (2) dentes dolente o maxillalgia al eveliar se, (3) restauratione frangite sin explication evidente, e (4) resorption ossee alveolar.

Le diagnose de bruxismo non es facile. Un grande numero de patientes suffre de illo sin saper lo. In tal casos on pote explicar al patiente que multe personas ha habitudes que illes ignora. Per acceptar le rolo de un observator impartial e vigilante le patiente pote possibilemente succeder a comprender consciemente le habitudes que ille cultiva inconsciemente. Le habitude de strider le dentes resulta usualmente in un destruction del texitos periodontal.

In le passato le tractamento de bruxismo esseva concentrate super su aspectos mechanic. Le therapia traditional consiste in restaurar le dimensiones vertical del dentes per varie medios mechanic. Le equilibration occlusional es un altere tractamento e debe de facto remaner un parte essential del therapia antibruxismal. Le construction de supportos servi a rememorar al patiente que ille non debe strider su dentes e a eliminar le punctos de interferentia.

Methodos psychotherapeutic deberea occupar un placia importante in le tractamento de bruxismo. Le analyse del historia del patiente, le uso de medios de autosuggestion, e le consultation de un psychiatro pote esser indicate in certe casos. In le majoritate del casos iste affliction pote esser considerate como le reflexion de un profunde perturbation emotional. Le alleviamento que resulta del elimination del tension emotional sequite per le correction del habitudes occlusional es multo satisfacente. Le arte del dentista, si illo vole justificar se como servitor del sanitate human, debe esser preste a recognoscer le possibilitate de un etiologia psychic e non solo physic o physiochimic in disordines dental. Per consequente nos debe effortiar nos usque al limite de nostre possibilitates a acquirer le cognoscentias necessari pro comprender le relation inter le processos psychic e somatic in sanitate e morbo dental.

MID-WINTER MEETING

AMERICAN ACADEMY OF DENTAL MEDICINE

Sunday, December 5th, 1954

HOTEL STATLER, NEW YORK CITY

THE ROLE OF THE DENTIST IN DIABETES MELLITUS*

Anthony Sindoni, Jr., M.D.†

The question—how can the dentist help or give aid to the physician in his treatment of diabetes mellitus as well as help discover the two or more million unknown diabetics—may first sound confusing. But when one realizes that in recent years the accomplishments in dentistry have revealed that this scientific field has made multiple strides not only in oral and dental diseases but also in preventive dentistry in cooperation with the medical profession, the question becomes simpler. This cooperation of both professions was revealed in the fluoridation of the public water supplies. As your past president of the American Dental Association, Dr. Fitzgerald so aptly claims: "one of the greatest advances in the history of preventive dentistry"[1] is an excellent achievement. The program, I understand, is in operation in over 800 communities. In other words one of every 9 persons in the United States is routinely drinking fluoridated water.

It is evident that dentistry is not only concerned in tooth repair or replacement but also with the condition of the supporting structural tissues of the teeth as well as with the state of the oral cavity. Thus such diseases of the oral cavity caused by drugs, such as bismuth, mercury, antibiotics; the infectious, membranous and catarrhal types of stomatitis; Vincent's infection, leukoplakia, cancer, syphilis, infections arising from the teeth and affecting the jaws, maxillary sinus and lymph nodes, and other diseases associated with dermatological, metabolic and nutritional conditions, may affect the whole body functions and often may be the initial lesion or inkling of an existing systemic disease.

Before discussing the close association of oral disturbances with diabetes mellitus I believe a general knowledge of the symptoms and signs of the disease will enable you to interpret more intelligently the dental or oral diseases associated with diabetes.

When one observes the increased incidence of diabetes mellitus throughout the world and its role in the life of the affected individual, he cannot help discussing the seriousness of the situation. This becomes more real than apparent when he realizes that the disease now takes an increasing death toll of the people of a community, city, state and country.

Thus diabetes as a cause of death in the United States has risen from twenty-seventh place in 1900 to tenth in 1949.[2] Report from the Metropolitan Life Insurance Company industrial experience in 1949 revealed that it ranks sixth as a cause of death transcending pneumonia and tuberculosis.[2]

As based upon statistical observations it is anticipated that within another five year period, including associated vascular diseases such as cerebral, cardiac and renal deaths, diabetes will rank third as a cause of death for the entire population of the United States.[2] This is readily recognized from the observation of the cause of death from coma compared with cardio-renal-vascular diseases in the pre and post insulin periods. Thus the cause of death from coma in 1898 to May 31, 1914 (pre-insulin era) was 63.8%, whereas from cardio-renal-vascular disease it was 17.5%; from January 1, 1944 to April 27, 1851 (insulin—Charles H. Best era) deaths from coma was 1.8% while the deaths from cardio-renal-vascular diseases had risen to 70.8%. As based upon the present mortality rate, it is predicted that by 1980 if the mortality continues to rise, deaths from diabetes will be surpassed only by those deaths from heart disease.

To a measurable degree insulin has conquered coma and increased the life expectancy of a diabetic, but our circulatory complications as previously mentioned are graver—affecting the usefulness of man either partially or completely as well as taking his life. These vascular complications affect the most vital parts of a diabetic's body—his eyes, heart, kidneys and lower extremities.[3]

In our own metabolic ward at Philadelphia General Hospital, a city supported institution,

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where the patients of the lower income group are cared for, we admitted 476 patients for the year of 1953, 122 or more than one quarter of whom suffered from gangrene or ulcer of the lower extremities.

The true etiology of these vascular conditions associated with diabetes is still unknown. There are many theories advanced by the various schools of thought to help substantiate their clinical findings of the close association of vascular degeneration to the control of diabetes and diet. Since this is a controversial issue, I can say only that I am still in a quandary concerning the true cause of the associated vascular diseases in diabetes.

I am cognizant of the fact that with increased incidence of diabetes in all classes of people throughout the world that diabetic coma is no longer a problem in the presence of insulin therapy. But the real issue that faces the individual with diabetes today is vascular complications.[4] How can it best be controlled? This is our challenge.

You members of the American Dental Association making a total close to 100,000 can play a vital role in this branch of medicine, first, by helping to discover the 2 or over 3 or more millions of living undiscovered diabetics in America; secondly, to caring for the diabetic oral cavity in co-ordination with the physician. The discovery of the unknown diabetic may be made when the following facts are evaluated:

1. *Careful diabetes-dental history:* Diabetes-Dental History should be incorporated in all dental offices as a routine procedure. The evaluation of the oral cavity cannot be determined alone without taking into consideration the general physical condition of the patient.

If a patient has a family history of diabetes or presents symptoms or signs of diabetes and he is not under a physician's care either one of the common or simple laboratory urine tests for sugar be made or the patient referred to a doctor for discussion of your findings, ought to be the rule. It is also the duty of the dentist to know whether the patient having diabetes is adequately following his physician's instructions.

2. *Factors involved in the etiology of diabetes mellitus:* The consensus of opinion based upon laboratory and clinical findings is that the cause of diabetes is associated with a deficiency of insulin. Whatever the etiology may be it is still unknown. We do know that the islets of the pancreas secrete insulin. Whether this is quantitatively or qualitatively deficient or neutralized by some other factors or factor unknown to us after its normal secretion by the pancreas is still a mystery. The fact remains, however, that whatever the various processes that affect the insulin mechanism the end result is insulin deficiency.

We may therefore conclude that whatever affects the insulin mechanism, the condition diabetes, is the result of insulin deficiency. Any person with a family history or some blood relationship having diabetes should be strongly suspected and examined for the condition. Heredity predisposition is considered of marked importance. A susceptibility to diabetes appears to be inherited as a Mendelian recessive characteristic. It may be revealed in the immediate member of the family or in a distant relative and may not appear until late in life. This becomes more apparent when you realize that it has been estimated that approximately one out of every 4 persons is considered to be a "diabetes-carrier."[5]

Obesity appears to be a very significant factor in diabetes when you observe that a large percentage perhaps 40-80% of the persons with diabetes were originally overweight.

Associated with obesity we may mention overeating. Alone overeating is not to be considered a potent etiology when you realize that insulin stimulates appetite. Perhaps these individuals over a long period of overeating may "exhaust" these islets of Langerhans producing insulin and develop diabetes.

In conjunction with obesity, recent studies have revealed the relationship of the endocrine glands—pituitary, adrenals, thyroid in the causal relationship of diabetes. When you realize the increased incidence of diabetes in obesity, and in overweight children you can not help thinking of the endocrine.

Other factors that appear to be playing a

significant role in diabetes are gall-bladder or biliary diseases and trauma. Its increased prevalence in patients past middle life and in women, and, as observed by some investigators, in those patients who have been on increased fat diets.

3. *Symptoms and Signs of Diabetes:* These play a role in exposing the presence of diabetes in a large majority of cases. The most characteristic symptoms are increased thirst, urination and appetite. These symptoms are usually accompanied by a gradual or rapid loss of weight and a general weakness. Other symptoms by the patient of which he frequently complains are pruritis, local or general, disturbed vision, "heavy" sensation, or tiredness, numbness and tingling sensation in lower extremities; suspectibility of skin to delayed healing ulcers, carbuncles or furuncles.

4. *Practical laboratory examination for urine sugar:* The simple or the popular equipment for laboratory examination of urine for sugar content should be routine equipment in all dentist offices.

The Benedict Qualitative, Sheftel, (urine sugar test case) Gelatest-Method (Denver Chemical Manufacturing Company) and Clinitest Table Reagent Method are the most common commercially available and inexpensive way of determining the sugar content in the urine which any intelligent layman can do without resorting to self diagnosis.[6]

These tests are time saving. The value of these urine examinations is increased when you realize that there are many persons who present no significant symptoms or signs of diabetes. Since patients are taught to examine their own urine there is no reason why you should not insist if the patient has no family doctor and conditions warrant it. When urine is positive, the patient should be referred to a physician for discussion of his findings. The fact that diabetes is present is important to the dentist's care of the oral cavity. He can adjust his therapy of the gums or oral lesions, extraction of teeth, replacement of teeth or denture according to the patient's response to diabetic therapy.

5. *The most common oral lesions associated with diabetes.* The mucous membranes of the oral cavity are generally sensitive indicators of some general physical disorder, whereas diseases of the oral cavity may have a pronounced effect upon the body functions. These effects are readily understandable when you realize the condition of the oral mucous membranes are influenced to a measurable degree by the same physiologic processes as are other parts of the body.

The oral lesions may often present suspicious signs or inkling of a metabolic, nutritional or endocrine disorders which may be associated with uncontrolled diabetes, but which are not specific for the disease diabetes. We are cognizant of the many disturbances created by uncontrolled diabetes especially when we realize that the consensus of opinion today is that diabetes mellitus is considered to be a syndrome or symptom complex, with a varied etiological background characterized by impairment of the body's normal ability to metabolize or utilize food.[4] This impairment is dependent upon the condition of the insulin mechanism—an actual or relative deficiency of insulin brought on by a disturbance in the function of the islets of Langerhans of the pancreas, or by some interference with the action of insulin in the body tissues. True insulin is concerned with the carbohydrate metabolism, but also with that of protein and fat.

Thus one can readily understand why such signs and symptoms of the oral cavity as dryness of the mouth, hyperemic and swollen gums and mucous membranes with a burning sensation of lips, tongue and palate, loss of filiform papillae on the tongue may be signs of avitaminosis frequently associated with uncontrolled diabetes. They may well represent oral manifestation of vitamin B complex deficiency.[7]

Observations have revealed that the high incidence of caries in diabetic patients is no greater than in the non-diabetic.[2] A careful dental study at a diabetic camp for boys and girls showed that among 276 children aged six to seventeen years 15.2 percent did not require dental care. It also revealed no increased incidence of missing, filled or carious teeth and of periodontal disease than that of a control group of non-diabetic children of comparable ages.[2] Another

group of investigators[8] in their studies of the oral cavity in 149 treated diabetics found the subjective symptoms and their tissue changes, "as denture irritation, dryness of the mouth, loose and painful teeth, burning lips, tongue and palate, receding, bleeding and painful gums, tenderness at temporomanidibular joint, loose and poorly fitting dentures, bad mouth odors, swelling in the parotid region. indentation, fissuring, lobulation of the tongue and hypertrophy of the papillae; for the gingivae: edema, recession, hypertrophy, bleeding, pus formation."

According to Cohen[9] the absence of increased salivary glucose levels in certain diabetics having the highest susceptibility to caries points to other factors in the physiology which may be involved in diabetes. His studies of an extended period for caries susceptibility of 27 young diabetics, aged 6 to 30 years observed an accelerated rate of tooth decay with advancing age.

In view of the above observations as well as of the results of other clinical investigators it appears that there are no specific oral signs or symptoms of diabetes. Similarly, a physical examination of a diabetic may too not show any specific signs of diabetes.

Perhaps Burket's[7] suggestive signs and symptoms as (1) patient's complaint of a tooth ache in an otherwise clinically sound teeth, (2) increase number of new cavity formation during a given period of time; (3) periodontal disease as a possible early sign; (4) development of advanced periodontal disease with marked loss of the alveolar bone in a young patient; (5) an unusually rapid calculus formation in uncontrolled diabetes; may add much to help the dentist make diagnosis of suspicious diabetes. With such symptoms or signs he should not hesitate immediately to consult the family's physician. Such cooperation will help give invaluable aid to the patient in helping to discover the disease in an early stage and perhaps in warding off some of the unfavorable complications.

ORAL CARE OF THE DIABETIC PATIENT

No physical and laboratory examination of the patient is complete without a thorough investigation of the oral cavity. It is the duty of the physician to advocate dental consultation as a routine procedure for all his patients. Since an adequate diet is a paramount factor in the treatment of a patient with diabetes, oral disturbances must be corrected to insure the proper mastication of food, aid its normal absorption and utilization, thereby helping to avoid avitaminosis. This latter aim we must try to attain.

It is the common belief among many diabetics that when their blood sugar level is normal the diabetes is adequately controlled. But clinical observations have revealed that, in spite of the normal blood sugar level, many of the patients have evidence of associated progressive vascular degeneration such as retinopathy, coronary artery disease, or advanced renal disease (Kimmelsteil-Wilson).[4] What the etiology may be is still unknown. The fact remains, however, that the blood sugar level should not be the sole reliable index to the state of whole body metabolism without considering, or searching for, other unknown factors.

A diabetic patient with a normal blood sugar level may have many oral disorders associated with diet disturbances. Thus it is not uncommon to have such patients complain of symptoms and signs of generalized avitaminosis such as swollen, hyperemic, tender gums, which bleed easily; loose and sensitive teeth with multiple cavity formation; enlarged, raw-like appearance of the tongue with soreness; general malaise associated with tiredness, mental depression, irritability; gastro-intestinal symptoms such as "bloated" feeling in the stomach, "heart burn," constipation or loose stools, as well as impaired vision, itchy, scaly and swollen eye lids."

Even after correction of the mouth disorders, by removal of all foci of infections and the replacement with proper dental structure to insure proper occlusion and normal mastication of the prescribed diet, there often results alleviation of many of the symptoms and signs of avitaminosis. As a result there is often a better control of the diabetes as evidenced by a reduction in the insulin dosage and sometimes its discontinuance.[12]

SURGICAL (ORAL) CARE

The oral surgical care, as extraction of the teeth in the person with diabetes is similar to that of a non-diabetic. It is in the unfortunate person who is unaware of his diabetes or who has uncontrolled diabetes where complications such as acidosis or coma, delayed healing processes or localized osteitis may develop or may be precipitated by the extraction of teeth.

That such complications do occur is sufficient evidence for the dentist to undertake all preoperative available measures for adequate oral care of the diabetic in co-operation with the family physician. These can be accomplished in the following manner.

First,—be certain the diabetes is adequately controlled. It is not uncommon for the patient to inform the dentist his diabetes is stabilized. This information may be true, but, rely on it only after you have consulted his family physician.

Second,—make a careful oral examination and decide upon the number of teeth to be extracted. Consult the family physician as to the patient's general physical condition to ascertain the optimum time for dental extraction.

It is the consensus of opinion and advocated by Burket and others[8, 10] that oral foci of infection such as pulpless or so-called "dead" teeth and periodontal disease (pyorrhea) should be given utmost consideration prior to surgery. One can readily understand the cause of increased transient bacteremias in diabetes following surgical intervention, if proper preoperative care is ignored. Therefore, if there are signs and symptoms of periodontal disease, it should be treated to help reduce the increased susceptibility to bacteriemias as well as the severity of the diabetes.

It is not uncommon to observe patients with uncontrolled diabetes suffering from periodontal abscess or diseased gums which are often separated from the roots of the teeth, which are loose or movable, with increased cervical sensitivity, which are fertile fields of transient bacteriemias. With the correction of the infection and the control of the diabetes, many of the oral changes become less pronounced, the teeth become less sensitive and less movable; gums approach the normal state in color and consistency and the tongue appears normal with the absence of mouth odor of acetone.

Oral surgical procedures are generally performed in the morning 2-3 hours after breakfast and insulin, using local infiltration or block anesthesia such as procaine hydrochloride, or lidocaine.[7] Substances or agents containing adrenalin or cobefrin are inadvisable because of the possibility of precipitating anginal symptoms or local adverse tissue changes.

If the condition of the oral cavity is such that the use of local anesthesia may help spread the infection, then it is advisable to consult the family physician for the type and use of a general anesthesia. The one generally employed for short or quick operation is nitrous oxide-oxygen. After the type of general anesthetic is decided upon, it is advisable to administer it to the patient in a fasting state, either before breakfast or four hours after breakfast.

When the patient gains consciousness immediately following the use of the general anesthetic no food or fluid is given orally for 1-2 hours. After this interval, the patient is given ginger ale, strained orange juice, followed by the regular dose of insulin until the next meal, when liquid or soft diet is given according to the diabetic formula.

It is not uncommon to observe signs and symptoms of hypoglycemia reactions 2 or more hours after tooth extraction in patients who have been administered insulin. This condition is frequently recognized by blurred vision, impairment of speech, profuse sweating, twitching of facial muscles and extremities, or even by unconsciousness (insulin coma). The condition is generally corrected by giving the patient ginger ale or some form of sugar, plain or dissolved in water or in orange juice. If the patient is in insulin coma intravenous administration of glucose is advisable.

The question of the use of antibiotics after extraction of teeth has been frequently discussed. Penicillin and other antibiotics are strongly advocated for patients following tooth extraction, especially in the presence of such definite infections as rheumatic, congenital and arteriosclerotic heart disease. The question of the ap-

plication of universal chemoprophylaxis must be thoroughly studied, especially when one observes the rarity of secondary bacterial infections following dental extraction and the increasing frequency of unfavorable or even fatal reactions to antibiotics.[12] It is our belief that the use of antibiotics be best restricted to specific conditions.

SUMMARY

1. The dentist's better understanding of diabetes will enable him to help discover 2 or more million unknown diabetics when the following facts are known.

(a) Diabetes-Dental History.

(b) Factors involved in the etiology of diabetes mellitus.

(c) Symptoms and signs of diabetes.

(d) The use of one of the common commercial inexpensive and available laboratory apparatus for examination for urine sugar in those patients with a history or suspicious oral signs and symptoms of diabetes not referred by doctors, or who are not under medical care.

(e) Common oral lesions or symptoms such as periodontal disease especially in the young with marked loss of the alveolar bone, rapid calculus formation, dry mouth, enlarged beefy red tongue with possible faint acetone breath, increased number of cavity formations in a given period of time and tooth ache in a clinically sound tooth are signs of possible diabetes.

2. Role of the mouth in health of patient.

(a) Normal conditions of gingivae, good teeth and normal occlusion to insure proper mastication aid in absorption and utilization of food, help avoid general avitaminosis, and result in better control of diabetes.

(b) Periodic oral consultation.

3. Oral care of the diabetic patient requires co-operation with family physician.

4. Oral surgical therapy includes:

(a) adequate pre-operative prophylaxis.

(b) type of anesthesia.

(c) optimum time for surgical procedures.

(d) the role of antibiotics.

(e) restoration of oral structures—dental replacements to approach normal occlusion for proper mastication.

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LE ROLO DEL DENTISTA IN DIABETE MELLITE

Dr. med. Anthony Sindoni (Filio)

SUMMARIO IN INTERLINGUA

(1) Un melior comprension de diabete rende le dentista capace a contribuer al discoperta del duo o tres milliones de non-recognoscite diabeticos in le Statos Unite. Pro isto le dentista debe esser familiar con le sequente factores:

(a) Le historia diabetic del patiente dental.

(b) Le factores involvite in le etiologia de diabete mellite.

(c) Le manipulation de un del varie non-costose e currentemente disponibile apparaturas commercial pro le detection de glycosuria in patientes con un historia de suspecte signos oral de diabete le quales ha recipite nulle attention medical.

(d) Le possibile symptomas e signos de diabete. Istos include: Lesiones oral commun o symptomas de morbo periodontal que es associate—specialmente in le caso de juvene patientes—con un perdita pronunciate de osso alveolar; le rapide formation de calculo; siccitate del bucca; allargate lingua carnicolor con possibilemente un exhalation levemente acetonic; un augmentate numero de cavitates formate in un definite periodo de tempore; dentalgia in dentes clinicamente non-morbose.

(2) Le rolo del bucca in le sanitate del patiente.

(a) Un condition normal del gingivas e bon dentes con occlusion normal e le resultante mastication adequate es de adjuta in le absorption e utilisation del alimentos. Illos contribue a evitar avitaminosis general e effectua un melior controlo de diabete.

(b) Consultationes oral periodic.

(3) Le tractamento oral de diabeticos require le cooperation del medico de familia.

(4) Le chirurgia therapeutic del bucca include:

(a) adequate prophylaxis pre-operative;

(b) typos de anesthesia;

(c) selection del melior tempore pro le intervention chirurgic;

(d) le rolo del antibioticos;

(e) restauration del structura oral—replaciamentos dental pro approximar le occlusion normal e assi un mastication adequate.

Medical Views on Oral Problems

DISCOLORATION OF THE TONGUE*

TO THE EDITOR:—*What is the significance of a brown, not black, tongue and also of one yellowish-tan in color? The yellowish-tan tongue I see in a large number of patients. It occurs on the central dorsal surface. The dark brown tongue, I have seen only about four times.*

H. Edward Hengen, M.D., Pattonville, Mo.

ANSWER.—A yellow to brown discoloration of the dorsum of the tongue is generally due to the staining of a previously coated tongue by certain foods, drugs, dyes, tobacco, or possibly chromogenic organisms. The type and intensity of the color depends on the staining agent and the duration of its use. Coating of the tongue results from a failure of the normal cleansing action of mastication and salivation to remove desquamated cells and other debris from the surface. Such factors as dehydration, gastrointestinal disease, and impaired mastication most frequently produce this condition. Brownish discoloration of the central third of the dorsum of the tongue is frequently associated with hypertrophy and matting of the filiform papillae. In addition to the factors already mentioned the following should also be considered: (1) the oral use of penicillin and other agents in troches and sodium perborate and (2) the systemic administration of antibiotics. There is apparently no direct relation between these conditions and nutritional disturbances. In some instances no specific cause is found. Further information is given in the chapter by Sarnat on "The Tongue in Health and Disease" in Portis' "Diseases of the Digestive System" (ed. 3, Philadelphia, Lea & Febiger, 1953).

* Queries and Minor Notes, J.A.M.A. July 3, 1954, p. 945.

TRIGEMINAL NEURALGIA: PATHOLOGY AND TREATMENT*

In trigeminal neuralgia (facial tic, or tic douloureux), the pain is spasmodic, and occurs in lightning flashes that usually are momentary but occasionally last a few minutes. In severe cases, the pain may recur many times a day. It is initiated by any slight movement of the face, as in chewing or speaking and a light touch may bring on an attack during which the patient remains completely still, often holding a hand near his face, as if for protection. This unwillingness to touch the painful area is of diagnostic significance, since in other types of facial pain the patient usually rubs his face to get relief. The pain commonly originates from a trigger point. No definite pathological change in the trigeminal pathways or the contiguous tissues is found to account for the pain. Organic lesions, such as neuromas and aneurysms, can cause pain indistinguishable from that of trigeminal neuralgia, but in most cases no such lesion is present. Histological studies suggest that the lesion is a "perifilamentous adhesive neuritis." Operations were performed in 176 of 250 cases observed by the author, who believes that it is wrong to render the whole face numb when only one area is painful. Only 34 of his 250 patients had pain in all divisions, and 172 had no pain in division 1. Spread of pain to division 1 is by no means inevitable. The author comments on the advantage of ganglion injection over complete root resection and on the advantages of fractional root resection in suitable cases. The intradural approach, though now safe in most hands, carries a greater risk to life than the extradural approach. Intracranial extradural neurectomy of the second division is a valuable procedure in suitable cases, since the territory of only the maxillary division of the face need be denervated when the pain is confined to this area. Occasionally, when doing a fractional root section, the author also does neurectomy of the second division when it is essential that no fibers of this division be spared, but no fibers of the first division sacrificed. With neurectomy of the second division alone, there is no danger of paralytic interstitial keratitis or of facial paralysis. Extracranial alcohol blocks of the second and third divisions are safe and produce local denervation only, but the relief is only temporary. Avulsion of the supraorbital or infraorbital nerve is worth trying in old persons when the trigger point seems to be in one of these areas. The relief gained, however, is merely temporary. Procaine injection into the gasserian ganglion will often bring to an end a bout of trigeminal neuralgia. Fractional root section by the middle fossa extradural approach is the treatment of choice in cases in which the ophthalmic division is not involved.

* Medical Literature Abstracts, J.A.M.A., July 31, 1954, p. 1286.

SCURVY OCCURRING IN PATIENT ON AN ULCER DIET: REPORT OF CASE*

The case presented concerned a man, aged 31, who in October, 1953, attended a Glascow infirmary complaining of a purpuric eruption of the legs of three to four months' duration. Examination revealed characteristic clinical features of scurvy; his skin

was dry and rough, and petechiae and ecchymoses were present on the legs and arms. The gums were bluish, hypertrophied, and infected; the teeth were carious and breath foul. In March, 1953, he had undergone a laparotomy for a perforated duodenal ulcer. After discharge from hospital, he suffered intermittently from postprandial epigastric pain. A bland diet was prescribed, but gradually the patient eliminated various items such as fruit and vegetables because he believed they precipitated ulcer symptoms. The patient was hospitalized. Capillary fragility was shown by positive Hess test. A vitamin C saturation test was carried out. While 700 mg. of synthetic vitamin C was given daily by mouth, urinary saturation was not detected until the seventh day. After saturation had been achieved, the daily dose of vitamin C was reduced to 100 mg. A patient with peptic ulcer is singularly apt to require emergency surgery, and in such an event the vitamin C reserves may be of great importance. The quantity of vitamin C available in diets is variable; opinion is unanimous, however, that 100 mg. of synthetic vitamin C daily will maintain tissue saturation. This is a convenient amount to prescribe routinely in all cases of peptic ulcer treated by dietotherapy.

* Medical Literature Abstracts, J.A.M.A., July 17, p. 1104.

MONILIASIS OF ORAL MUCOUS MEMBRANE*

The occurrence of moniliasis of the tongue and of the hard palate is described in a 51-year-old woman. Greasy ulcers, with a whitish coat, very tender to touch, were present for 32 months at the tip of the tongue, at the soft and hard palate, and on the mucosa of the vestibule of the mouth. The condition was kept stationary with local application of various disinfectants and administration of 3,600,000 units of penicillin. Some of the ulcers were removed by curet with the aid of hexobarbital sodium anesthesia. Repeated smears of the floor of these ulcers revealed profuse growth of Candida albicans. The bacteriological diagnosis of C. albicans was established by sugar fermentation tests. Fermentation of dextrose, levulose, and maltose was produced, but fermentation of saccharose and lactose did not occur. The causative role of C. albicans was demonstrated by the hemagglutination between the serum of a rabbit (which had been infected with the strain obtained from the patient) and the erythrocytes of the patient. Temporary improvement was obtained with local application of 25% glycerin borax and a pyrazol derivative combined with oral administration of 60 gm. of a proprietary sulfonamide compound. Best results were obtained with daily local applications of malachite green for eight weeks, combined with quartz lamp irradiation and administration of tonics. The clinical aspect and the bacteriological and serologic findings justified the diagnosis of moniliasis. Prognosis for cure is unfavorable, and prognosis for survival is doubtful. There is no effective chemotherapy available for moniliasis. Ingestion of food will be difficult temporarily and will lead to loss of weight and strength and consequently to increased tendency to infection. Fatal invasion of the circulation must be expected eventually.

* Medical Literature Abstracts, J.A.M.A., June 19, 1954, p. 793.

PREVENTION OF DENTAL DECAY*

In May, 1945, the Child Dental Health Committee of the British Dental Association reported on the dental health of children to the Minister of Health, Minister of Education, and other public bodies. A national campaign to prevent dental decay in children instead of using costly and ineffective methods of restoring and replacing diseased teeth is recommended. The committee urges testing the value of fluoridating water supplies as a means of reducing dental caries in children. Mr. Stewart Ross, chairman of the Council of the International Dental Federation, commented at a press conference on May 3 that American experience showed that fluoridation of water reduced the incidence of dental caries in children by about half. It should be possible to make a controlled test in Britain because North Shields had an adequate supply of fluorine in the drinking water, whereas South Shields did not. If fluorine was an important factor in preventing dental decay, the effect should be seen by observing the incidence of dental decay in the two towns. The committee also recommends that the school dental service be transferred from the local authority to the Ministry of Health. School children are at present receiving inadequate dental care because of the difficulty of obtaining school dentists, who are inadequately paid by the local authority. Parents and teachers should be warned of the bad habit of giving children sweets between meals. This, and the giving of sweets, biscuits, and sweetened liquids at bedtime, is considered to be a potent factor in causing dental decay. In Britain only one person in 50 has sound teeth, and it is estimated that only 17 million toothbrushes are bought a year by a population of 50 million.

* Foreign Letter, J.A.M.A., July 3, 1954, p. 923.

SYSTEMIC LUPUS ERYTHEMATOSUS: EARLY CYTOLOGIC DIAGNOSIS*

According to Dubois, systemic lupus erythematosus is relatively common. At the Los Angeles County General Hospital, 44 new cases were diagnosed in 1950 and 1951, as against 88 cases of acute rheumatic fever. Only 18 cases of Hodgkin's disease, 39 cases of acute and subacute leukemia, and 36 cases of pernicious anemia in relapse were observed during

this same period. The diagnosis of systemic lupus erythematosus is difficult because of the variable nature of the symptoms, which resemble those of many other conditions. In 1948, Hargraves described the characteristic cell of lupus erythematosus (L. E. cells), and since the introduction of Hargraves' test, which seems to be pathognomonic, it is usually possible to confirm a tentative diagnosis of systemic lupus erythematosus. Dubois describes his own simplified technique for the detection of L. E. cells. In every case in which the disease is suspected, two tests should be made of peripheral blood and one on aspirate from bone marrow, for sometimes the characteristic cells may be found on only one of these tests. After separation of a heparinized sample by centrifuge, a drop from the buffy coat is Wright-stained on a slide and examined for rouleaux formation and for a hematoxylin-staining material sometimes seen in inter-cellular bodies (which may be surrounded by a rosette of leukocytes) and sometimes seen as ingested by a leukocyte. Only the last finding is positively diagnostic of lupus erythematosus. A statistical analysis of 62 cases treated at the Los Angeles County General Hospital is given. Because of the frequency of rheumatoid-like arthritic changes in the disease, all patients with this form of arthritis should be given the test. Spontaneous remission and then relapse after a long asymptomatic interval occurred in many cases. With early diagnosis and vigorous treatment with cortisone and corticotropin, many patients can be relieved of symptoms.

* Medical Literature Abstracts, J.A.M.A., June 26, 1954, p. 864.

INTESTINAL TOXEMIA AND PERIODONTITIS*

One hundred patients with periodontal disease were tested for indicanuria. Ninety-eight showed positive findings. Of these twenty-four were 3-4 plus, 55 were 2 plus, and 19 were 1 plus. One hundred students tested at random showed twenty-three positive findings; eight were three or four plus, four were 2 plus, eleven were 1 plus. Ten of the twelve with high indican were subsequently examined. Seven had marked generalized, or moderate gingivitis. Of the 77 students who were indican free, seven had marked or moderate gingivitis.

* By Paul J. Boyens, D.D.S., Dental Digest, pp. 223-4, May 1954. Summarized by Arthur N. Yohai.

IS THERE A THERAPEUTIC DENTIFRICE?*

The editors point out that there is no "therapeutic" dentifrice; at best the effect sought is prophylactic. The general use of penicillin in dentifrices is not advised. Clinical studies of school children using dibasic ammonium-phosphate-urea dentifrice failed to show significant caries reduction after two years. There is no evidence that "chlorophyllins" will prevent caries or periodontal disease. The claims for "de-odoring" are grossly exaggerated. Clinical studies of "anti-enzyme" dentifrices are in the preliminary stage.

* Editorial, J.A.M.A.; Vol. 155, No. 4, p. 366, May 22, 1954. Summarized by Arthur N. Yohai.

THERAPY OF SYNCOPE*

Vasodepressor syncope is the most common encountered by physician and dentist, especially the latter. Fainting occurs because, in the upright position, pooling of blood occurs primarily in the lower extremities due to vasodilatation in muscle. Most effective therapy is not in the head-between-the-knees position, but rather the Trendelenburg or the supine or the prone position with the legs raised. Where the danger of foreign body aspiration exists, the prone position is preferable; otherwise the other two are readily achieved in the dental chair.

* By Alfred Soffer, M.D., J.A.M.A., Vol. 154, No. 14, pp. 1177-9, April 3, 1954. Summarized by Arthur N. Yohai.

CORONARY DISEASE

Coronary diseases greatest incidence is in the early fifties, when men equipped by experience and training should yield their greatest contribution to industrial progress. Every case must be individually assessed, and you must steadily avoid the far too common and equally ignorant assertion that the manifestation of coronary disease marks the end of a man's useful working life. A man may do in safety anything he can do in comfort, and he is well advised to remain active within his limits of comfortable exertion, rather than sink into himself and lead a life of invalidism. If he remains active, I believe that the development of the collateral circulation is assisted by the increased blood flow which accompanies effort, whereas it is probably hindered by the stagnating blood flow of ill-advised rest.—U. L. Brown, Heart Disease, in Industry, *The Medical Journal of Australia*, Feb. 13, 1954.

THE USE OF RADIOISOTOPES AS TRACERS IN CIGARETTE SMOKE*

Methods were developed for entraining certain radioactive materials in smoke from cigarettes and for depositing this radioactive smoke in the lungs of anesthetized dogs. The pattern of the smoke deposit in the lungs was mapped by measurements with the Geiger-Müller tube and by radioautographic techniques. This paper describes the general method and gives results obtained with three radioactive isotopes, sodium (Na^{24}), potassium (K^{42}), and arsenic (As^{76}). More than 20 dogs were used in studying the pulmonary distribution of smoke from cigarettes containing K^{42}. Experiments on the first few animals

served to develop the standard technique. Sixteen dogs were carried through the standard smoking procedure with K^{42}. Taken as a whole, these experiments show a diffuse random distribution of smoke particles throughout all areas of the lung. Ten dogs were used in studying the pulmonary distribution of smoke from cigarettes treated with Na^{24}. Satisfactory results were obtained in 8 of these 10 dogs. The pattern of distribution was similar to that obtained with K^{42}; there was a diffuse random deposit of smoke throughout all areas of the lung. When A^{76} is employed as the tracer substance in cigarette smoke, the amount of radioactive material deposited in the lung is far greater than with the other substances, a fact that is due to the larger amount of arsenic entrained in the smoke. The use of radioisotopes as tracers in cigarette smoke has certain limitations.

* Medical Literature Abstracts, J.A.M.A., Aug. 21, 1954.

INTESTINAL POLYPOSIS WITH MELANIN SPOTS OF ORAL MUCOSA, LIPS, AND FINGERS*

Peutz of Hague described in 1921 a family in which several members had pigmented spots on the lips, mouth, hands, and feet. The characteristic pigmentation was associated with generalized intestinal polyposis in seven of these persons. The cases covered three generations, thus suggesting a familial tendency. Foster,[1] in 1944, reported the occurrence of adenocarcinoma of the small intestine in father and daughter, causing intussusception. Both patients had diffuse brownish pigmentation of the lips and face. The father's brother showed similar pigmentation, and he had been hospitalized in 1935 because of attacks of abdominal pain and the passage of blood and mucus from rectum. Touraine and Couder[2] in 1946 described an instance of a "syndrome of Peutz" under the heading of "periorificial lentiginosis with visceral polyposis." Their patient, a girl 14 years old, had the characteristic pigmentation described by Peutz and a large rectal polyp, which on microscopic examination proved to be an adenoma.

In the United States, Jeghers and his associates[3] have observed 10 cases of intestinal polyposis and melanin spots of the oral mucosa, lips, and digits. The characteristic pigmentation was observed from early childhood. All of the patients were of dark complexion. The most impressive feature in these cases was the consistent and pecular distribution of the pigmentation. It was most striking on the lips and buccal mucosa and occurred in round, oval, or irregular spots and patches of brown or, occasionally, almost black pigment. Some of the spots had a stippled appearance when examined with a high power magnifying glass. No patient showed pigment on any part of the body in addition to the areas noted. The nature of this pigment anomaly remains obscure. The second feature characterizing what appears to be a definite syndrome is the intestinal polyposis. The polyps are distributed throughout the entire intestinal tract, with their most striking clinical manifestations in the small intestine. The manifestations are abdominal pain and signs of minor obstruction terminating in one or more attacks of small intestine intussusception. Jeghers and his co-workers point out that the majority of the polyps, as revealed by operation or autopsy, were located in the small intestine, principally in the jejunum. Peutz had similar experience in his seven cases. The polyps on microscopic examination proved to be benign adenomas.

The syndrome appears to be inherited as a simple Mendelian dominant. Besides the 10 cases referred to above, these authors mentioned 3 other cases and reviewed 12 proved cases from the literature and 5 probable cases. Kitchin[4] reported in 1953 on two patients who came to the hospital with symptoms of intestinal obstruction due to intussusception. Both had the typical pigmentation in the oral region. Individual cases were reported by Kaplan,[5] by Perry,[6] and by Wolff.[7] Recently Savage[8] and Smith[9] each reported a case. The syndrome characterized by a characteristic pigmentation of the lips, oral mucosa, face, and digits, associated with multiple polyposis of the intestinal tract, with a strong tendency to familial incidence, appears to be a well-established clinical entity.

* Editorials and Comments, J.A.M.A., July 17, 1954, p. 1063.

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CARCINOMA OF THE FLOOR OF THE MOUTH

This paper is one of four on carcinoma of the floor of the mouth. The report is based on 118 patients observed at Westminster Hospital, London, from 1925 to 1951. Ninety-seven of these were treated before 1947, but eight of them were lost sight of in less than five years. Twenty-four of 89 suitable for assessment remained well for five or more years. Carcinoma of the floor of the mouth is a clinical entity only in its early stages; involvement of adjacent structures is common. The tumor is an epithelioma varying in keratinization and anaplasia. About 80% of these lesions occur in men. The differential diagnosis must rule out chronic inflammatory conditions, salivary calculi, and specific infections and in addition attention should be drawn to four other conditions that may cause difficulty in diagnosis: (1) benign mucous-and-salivary gland tumors primarily situated in the floor of the mouth; (2) acute monocytic leukemia with ulceration in the mouth and enlargement of the cervical lymph nodes; (3) metastasis in the lower jaw and adjoining gum and mouth floor from a clinically silent primary growth in the tongue; and (4) a primary malignant growth in the mouth that may be a sarcoma of the rhabdomyosarcoma type. A further point of interest in cancer of the mouth floor is the occurrence of a second and independent primary growth, sometimes many years after the appearance of the original lesion in the mouth. In the cases under review, such tumors occurred in the urinary bladder, rectum, bronchus, esophagus, or elsewhere in the mouth and pharynx. The tendency to familial occurrence was demonstrated by the occurrence in two sets of brothers (one of them twins) and in one set of sisters. Radiation therapy is the method of choice in early localized lesions. The author used mostly radium needles and teleradium, because neither intraoral radium applicators nor roentgen irradiation produced encouraging results. Local involvement of the tongue and gums require local diathermy in addition. Involvement of bone and widespread disease in the mouth are more amenable to extensive surgical excision. Except for bone grafting, the local closure should be accomplished as quickly as possible. Tube pedicles are badly tolerated by patients with mouth cancer, and other methods, such as rotation or sliding flaps, should be employed. In view of the magnitude of the surgical procedure, only patients with a good prognosis should be submitted to surgery. Criteria of inoperability are (1) spread posteriorly and involvement of the pharyngeal mucosa or the pharyngeal part of the tongue; (2) adherence of the lymph nodes to the vascular sheath; (3) edema of the subcutaneous tissue; and (4) gross involvement of the soft tissue of the submandibular and submental areas.

* Medical Literature Abstracts, J.A.M.A., Aug. 21, 1954, p. 1540.

CARCINOMA IN A NEWBORN: REPORT OF A CASE*

Occurrence of carcinoma of the cheek is reported in a newborn baby boy. When the infant was 2 days old, a rash was noted on his face and the attending nurse felt a lump in his left cheek. This lump increased decidedly in size over the next month. When examined at the age of 6 weeks, the infant was well nourished, extremely active, and completely normal except for the swollen left cheek. At operation a mass the size of a pigeon's egg occupying the substance of the cheek was freed from the skin and the mucosa intraorally and was removed. Microscopic examination of the specimen revealed a small cell carcinoma of salivary origin. There seemed to be uneventful recovery until three weeks after the operation when a left submaxillary node was felt. The next day the mass was removed en bloc. Microscopic examination showed that practically all of the lymph node had been replaced by a malignant growth consisting of hyperchromatic epithelial cells with large nuclei and nucleoli. There was a large number of mitotic figures. After three months, the infant's mother noted some blood-tinged saliva when he drooled. A cytological study revealed malignant cells, and a localized mass was widely excised. The microscopic report was that of a very cellular growth with considerable anisocytosis, anisonucleosis, hyperchromatism, and presence of large nucleoli and frequent mitoses. After the three surgical procedures performed when the infant was between the ages of 6 weeks and 5 months, the child developed normally and at 18 months he weighs over 33 lb. (14.5 kg.), is well, and is apparently free of tumor. The microscopic study suggested that the site of origin was probably the parotid gland. Differentiation of the parotids is not complete until some time after birth. The embryonal origin theory, therefore, seems to find substantiation in this case.

* Medical Literature Abstracts, J.A.M.A., Aug. 28, 1954, p. 1609.

Book Reviews

The Physician and His Practice, edited by Joseph Garland, M.D., Editor, New England Journal of Medicine with eighteen contributors; Little, Brown and Company, Boston, Massachusetts, 270 pages. Price $5.00.

The young professional man enters practice under great handicap. He is thrown into competition with those who have more experience from every practical angle and must impress his patients sufficiently to inspire their confidence while at the same time he must organize and operate a smoothly running office. The modern young practitioner's training is of the best, since all Medical and Dental schools have very high standards and their curricula are quite uniform in content and quality. The latest knowledge and techniques are in his possession. However, in order to apply these promptly to the advantage of the public, it is necessary that the novitiate either works with an experienced clinician or replaces such training by a formal guide to office management and professional and social deportment. By this means the tiro can avoid spending much time in developing office routine through stumbling experimentation.

For Dentistry the answer has come in the several books which are best exemplified by that of Blass and Tulkin.* In the text under review we find an admirable counterpart for the medical practitioner. Such texts are not only essential for the newcomer to office practice; they are almost equally important for the guidance and refreshment of practitioners of any age and experience.

The Physician and His Practice is a composite of the advice of authorities in many fields of Medical practice. It starts with the background of medicine, and discusses family and community relations and how the practitioner's wife can contribute to the success of his practice. It explains the opportunities in general medicine and specialty practice. Group practice, which this reviewer feels is the practice of the future, is given adequate coverage. Important auxiliary fundamental activities such as medical organizations, reading and writing of medical literature, community medical resources and the hospital are all given expert coverage. The chapters on Laboratory Facilities, Drugs and Medical Supplies, The Office and Related Business Aspects, Auxiliary Services, Office Records, Accounting Practices, Economic Security, and Medicine and The Law will prove practical and valuable as well as interesting reading.

Although this is primarily a physician's guide, it can serve the dentist well in his own practice and in his relations with his medical confreres, hospital, and other associated endeavors. It will also give the dentist a closer insight and a better understanding of the functioning of Medicine in its various categories of general practice, specialty, hospital and community services.

SAMUEL CHARLES MILLER

Essentials of Removable Partial Denture Prosthesis by Oliver C. Applegate, D.D.S., D.D.Sc., F.A.C.D., W. B. Saunders Co., Philadelphia and London, 1954, 352 illustrations, with 10 in color, $10.00.

Dr. Applegate's book fulfills a longtime need for a better text on a Partial Denture Prosthesis. The volume primarily teaches the principles of, outlines the planning for, and charts an efficient procedure in a design and construction of a removable dental prosthesis. The book is particularly directed toward the needs and problems of the clasp-retained appliance which depends upon the ridge tissue for its principal support.

Fundamental concepts are fully presented in order that procedures for actual construction may be taught not by dogmatic statements but through essential understanding. A great part of this book is devoted to step by step presentation of methods of construction for the effective prosthetic appliance. Many case history pictures illustrate the minute steps in careful planning and building. Much of the material in the book is presented in brief question and answer text and in detailed drawings and photographs of procedure.

The text is printed on glossy paper. Its page size is larger than the conventional text, but this is an asset rather than a liability. The larger than average type makes for easy reading. The pictures throughout the text are exceptionally clear and illustrate the points discussed.

This text is recommended for the dentist who desires a review of the subject of Partial Denture Prosthesis. It is a welcome addition to dental literature.

IRVING YUDKOFF

* Successful Dental Practice: J. L. Blass and I. Tulkin, J. B. Lippencott Company, Philadelphia, Pa., 1947.

Eleven Blue Men and Other Narratives of Medical Detection, by Berton Boueche, Little Brown and Co., Boston, 1954, 215 pages, $3.50.

This is not a textbook in the usual manner, but if more texts were written in this style, perhaps many more would be read. Berton Roueche is a member of the staff of the *New Yorker* magazine. This book is a series of twelve authentic stories of medical detection which first appeared, starting in 1947, in the *New Yorker*.

The detectives in each story are not police officers. They are physicians, medical inspectors, epidemiologists or research scientists. For the most part the criminals are microbes; and I dare say a better text describing all of the techniques, methods and fundamentals of bacteriology would be hard to come across.

In the story, The Alerting of Mr. Pomerantz, he describes the Koch postulates for the transmission of microbes as follows:

> The usual method of isolating a disease organism, and the one that Dr. Huebner followed, involves inoculating a group of highly susceptible laboratory animals whose reactions to disease are known to be similar to man's with a specimen of blood obtained from a person suffering from the disorder. Only if an animal becomes sick and exhibits the symptoms of the disease, as happens with exasperating infrequency in the case of so mild an affliction as rickettsialpox, is the transmission considered a success. The next step is simply a check to make certain that the results of the first were not a fluke. It consists of inoculating another group of animals with a fluid made from diseased brain tissue of infected animals. If any animals in the second group come down with the disease, the organism is presumed to have been isolated, and fluid made from the brain tissue of these animals is then injected into the yolk sac of fertile chicken eggs. There the organism multiply with exceptional alacrity, and thus a denser concentration of them is obtained. Smears of the yolk sac are next placed on slides, a very delicate operation, and at last, if everything has gone right, the organism becomes visible under the microscope and, with luck, identifiable.

I wonder how often the epidemiology of acute necrotizing ulcerative gingivitis has been proven false with *this* type of testing?

Throughout the stories several acrid remarks appear about the subject of diagnosis. In discussing the diagnosis of gout, and quoting a reliable authority on the disease, he says, "Some doctors appear to have managed to close their minds to the obvious almost indefinitely."

In another story about small pox and the embarassment of two physicians for not diagnosing several cases at a well known New York hospital for communicable diseases he writes, "They calmed themselves with the reflection that diagnostic infallibility is more often the aspiration than the achievement of any hospital staff."

In commenting on a physician's diagnosis of a disease that was at that time unknown he writes, "Dr. S..... closed his record of the case with the cautiously generalized though not inaccurate diagnostic notation, 'Sepsis with a rash.' Most physicians recover promptly from the distress of uncertainty. They are accustomed to it."

Readers of *The Journal of Dental Medicine* are very often called upon to see and diagnose patients with temporomandibular disturbances. Not too infrequent a patient will be unable to open the mouth because of a disturbance to the temporomandibular joint. True lock jaw caused by the Clostridium tetani elaborating the toxin causing tetanus is described as follows:

> Tetanus is the name generally preferred by physicians for lockjaw. They consider the latter more apt than adequate. The rigors of tetanus are by no means limited to an inability to move the jaw freely. Constriction of the masseter muscles is merely the characteristic symptom of onset. The physical manifestations of a full-fledged case of tetanus amply reflect the virulence of its cause. They are of such distinctive vigor and variety that the disease was among the first to be recognized as an entity. The accounts of clinical studies made by Hippocrates in the fourth century before Christ include one of a tetanus seizure that is still regarded as a model of acute observation. ". . . then trouble with his tongue, he complained he could not speak properly. The presence of tetanus was diagnosed, his jaws became pressed together, his teeth were locked, then symptoms appeared in his neck; on the third day opisthotonus appeared with sweating. Six days after the diagnosis was made he died." Opisthotonus is a spine-cracking muscular spasm.

This book should be read.

NATHAN WACHTEL

Oral Cancer, by J. Roy Bourgoyne, B.S., D.D.S., Chief of the Division of Oral Surgery, University of Tennessee, College of Dentistry and contributors; Lea and Febiger, Washington Square, Philadephia 6, Pa. 295 pages. Illustrated. $6.50.

The subject of this volume is so extremely important that the presence of another book on this theme is welcome. This is a fine summary and review of oral cancer. This book is recommended to dentists since it contains much fine and constructive material and is well grounded by adequate bibliographical references. The Glossary and sections on Methods of Cancer Diagnosis, Biopsy, Diet for Cancer Patients and Treatment of Cancer are especially worthwhile.

SAMUEL CHARLES MILLER

The Influence of Local Anaesthetics on Living Tissue in White Rat, by Gunnar Björlin; CWK Gleerup, Lund, Sweden. 112 pages, 34 tables and 13 illustrations.

This is a monograph concerning research carried out at the State Dental School of Malmö, Sweden. The effects on the tensile strength and healing qualities of the tissues of the white rat of procaine, monocaine and xylocaine were studied in a very thorough and scientific manner. Xylocaine was found to reduce the tensile strength of the healing tissues more than monocaine or procaine. The author cautions that in spite of his findings on laboratory animals, "No really deleterious effect of xylocaine and xylocaine-epinepherine has been observed during extensive use in oral surgery."

Björlin observed hyperemia in human tissues after the use of both procaine and xylocaine which he did not find in the white rat. The greatest injuries to the tissues in which the solutions were used appeared in the blood vessels. Xylocaine produced signs of necrobiotic changes in the arterioles after one day while less severe vascular changes revealed themselves after two days from the time of injection of procaine. Saline solutions used as controls showed practically no tissue changes.

SAMUEL CHARLES MILLER

Practical Full Denture Prosthesis, by Joseph S. Landa, D.D.S., F.A.C.D., Second edition. Dental Items of Interest Publishing Co., Inc., Brooklyn, N. Y., 504 pages, 218 illustrations, $9.50.

During the last few decades the life span of our population has been constantly increasing. This has created a vast reservoir of older patients which the dental practitioner must cope with. The dentist today must deal with many denture problems from anatomical, mechanical and psychological points of view. Dr. Landa's newly enlarged second edition will be of great help to the practitioner who is confronted with these problems because he thoroughly discusses and helps the reader eliminate the anatomical, mechanical and psychological difficulties.

The text is down to earth and avoids extravagant theories. It deals with the principles essential to the successful solution of some of the problems we are faced with in complete denture construction. The book emphasizes practical chair procedures and is aware of the needs of the dental practitioner. This edition has six new chapters which deal with frequently changing concepts in many phases of Prosthetic Dentistry.

The second edition has two contributors who are eminent exponents of denture prosthesis. The chapter on "Denture Problems in Complete Denture Prosthesis" is written by Dr. Raymond J. Nagle, Dean and Professor of Prosthetic Dentistry, New York University College of Dentistry. Dr. Clyde H. Schuyler, formerly Professor and Director of the Prosthetic Department at New York University College of Dentistry, has written a chapter entitled "Fundamental Principles in the Correction of Occlusal Disharmony, Natural and Artificial". With the aid of these two contributors, Dr. Landa has written a Prosthetic text that is a must for every dental library. The book discusses all the phases of full denture construction, rebasing, immediate dentures, and cleft palate cases. Of special note is the chapter on "Trouble Shooting". This chapter can save many dentists from mutilating new dentures because of their desire and over-eagerness to rid the patient of all his troubles at one time.

The book is printed on glossy paper and the illustrations are clear and accurate. There is an 11 page bibliography and a thorough index at the end of the book.

IRVING YUDKOFF

Doctor Dan—Pioneer in American Surgery by Helen Buckler, Atlantic, Little, Brown and Company, Boston, 1954, 381 pages, 7 illustrations, $5.00.

Writing with an almost religious intensity Miss Buckler tells an agreeable story of Dr. Daniel Hale Williams, better known to his patients and friends as Doctor Dan. A good deal of research, made possible by assistance from the Religious Society of Friends enabled the author to bring the work of this great Negro physician and surgeon from virtual obscurity to prominence.

The book begins with Daniel Williams' birth in 1856 and ends with his death in 1931 and although the events of his personal life and marriage seem somewhat two-dimensional, the accounts of his daring and resourcefulness as practitioner and surgeon are compelling and vivid. For it is in these areas that Miss Buckler tells the story, tersely and with technical soundness. It is too bad that in other sections of the book we are so constantly reminded of Dr. Dan's nobility and virtue, not so much by anecdote as by the biographer's insistence, that he emerges as something of a prig personally. Yet there is no question of his professional and sociological contributions.

Here was a man who might easily have passed as white, fair-skinned, red-haired and aquiline of feature, yet in times and places that were truly oppressive to the Negro professional he chose to identify himself with the small strain of Negro blood in his veins and to persevere in behalf of the people he had chosen as his own. With this to guide him he founded the first inter-racial hospital in Illinois, Provident of Chicago. He established there as well a Nurse-training Institute to which young Negro women might come and receive training equal to that of any other hospital. He was an intimate of President Cleveland. In 1893 he successfully sutured a torn pericardium and followed this historical piece of sur-

gery with other operations equally daring and bold. What then caused his fall into obscurity? Miss Buckler believes it to have been his disagreement with the policies of the Negro titan, Booker T. Washington. At any rate there is sufficient documentation at the end of the book to enable the reader to sift through and evaluate the facts.

G. PARKER

Histology edited by Roy O. Greep, Ph.D., with 13 contributors, P. Blakiston Co., New York, 1954, 648 illustrations, 953 pages, $15.00.

In the preface the editor reveals that this work, originally conceived as a sixth edition of Bremer and Weatherford, in reality became a new text.

This reviewer, while a student in school, studied histology from one of Bremer's earlier texts. It is apparent, when one compares the two works, that the presentation of this subject has come a long way in the last two decades. The manner of presentation has vastly improved, making this once tedious subject, a living, absorbing, and vitally interesting study. In addition the development and use of such media as the phase-contrast and electron microscope and improved techniques of preparing and staining tissues has markedly increased its scope.

The illustrations with few exceptions are photomicrographs of excellent quality clearly depicting the text description. Those diagrams which are included are mostly the work of Miss Etta Piotti, long recognized as an expert in her field.

Professor Greep has made an excellent choice of contributors for this work. Each chapter flows smoothly into the next although written by different authors.

It is difficult to cover all of the highlights of a book which includes 35 chapters and some 950 pages. For the dentist, the chapter on the Oral Cavity will be as informative as any he has read in a general histology text. It includes some embryology and histochemistry, and above all, excellent illustrations and descriptions of some of the elements of bone, enamel, dentin, cementum, periodontal membrane, etc., heretofore not clearly presented.

The remainder of the text deals with equally excellent information on the systems and organs of the body.

This book is highly recommended to those interested in relearning and acquiring new knowledge in a subject so vitally related to all branches and facets of living science.

ALLAN N. ARVINS

The Science of Dental Materials by Eugene W. Skinner, Ph.D., W. B. Saunders Co., Philadelphia, 1954, 456 pages, Illustrated, $7.50.

This edition of the already well known book brings the contents up to date and expands its coverage. Although the author states that it is intended as a textbook for the first year dental student and not as a reference, the material presented might very well be used as source of reference for the practitioner.

The dental materials generally used are fully discussed with their practical applications evident. Many of the materials commonly used in the dental office are grossly mishandled. A review of their physical properties would result in securing far greater utilization and hence conservation of time.

Reading the section on distortion of impression compound or on the use of hardening solutions for alginate impressions alone will convince the reader of the value of this book.

The chapter on tooth restorations with acrylic resins is well written. It includes a presentation of the several techniques for this type of restoration and reasons for possible failures are cited.

The American Dental Association specifications for dental materials are stated in the appendix.

Although this is not a reference book for the researcher, the general practitioner should include this textbook for very profitable reading.

GEORGE J. WITKIN

American Textbook of Operative Dentistry, by Arthur B. Gabel, A.B., D.D.S., M.A., and seven contributors, Lea and Febiger, 9th edition, 626 pages, 422 illustrations, $10.00.

Every text should recognize the limitations of its scope. The selection of boundaries has always proven difficult for most authors. This problem is well illustrated by the contents of this book.

The contributors range through many fields of dental education. Some of the subjects have been treated in textbooks as single entities. The subjects include "Examination and Diagnosis", Paul E. Boyle; "Caries", Philip Jay; "Preventive Dentistry", Russell W. Munting; "Metal Inlays", William D. Vehe; "Special Operative Procedure", Thomas J. Hill; and "Restorative Materials", Floyd A. Peyton. Two chapters by the author on "Operative Procedure" and "Mechanics" complete the book's content.

In the preface to this edition, Dr. Gabel emphasizes that this edition has been primarily intended for use by students. As a textbook, the least desirable feature is the inadequacies of the illustrations and the lack of color plates. It would be easier, for example, to visualize the different shades of a gas-air blowpipe flame, or to recognize periodontal lesions, if the illustrations were in color.

Pertinent material for effective instruction of students in operative dentistry is presented. The author is Professor of Operative Dentistry at the School of Dentistry, University of Pennsylvania. His long teaching experience is reflected in the concise and pertinent discussions found in this text. This book contains a tremendous amount of information, and is to be recommended for students and practitioners alike.

JULIUS N. OBIN

Academy Proceedings

MID-WINTER MEETING OF THE AMERICAN ACADEMY OF DENTAL MEDICINE

The American Academy of Dental Medicine will hold its annual Mid-Winter Meeting and Luncheon at the Hotel Statler in New York City, on Sunday, December 5th, 1954. There will be a business meeting for members at 10 A.M. followed by a luncheon. The afternoon will be devoted to a discussion on "Bone." The panel will be composed of both physicians and dentists. All members, interested dentists and physicians are cordially invited. The essayists for the afternoon will be:

HARRY SICHER, M.D.

Topic: Bone Formation and Resorption.

J. P. WEINMANN, M.D.

Topic: Variations in the Structure of Bone Tissue and Their Significance in Radiology.

LESTER CAHN, D.D.S.

Topic: The Correlation Between Clinical, Roentgenologic and Histologic Studies in Bone Lesions.

DR. ALEX SOBERMAN, *Co-Chairman*
DR. NORMAN E. ALDERMAN, *Chairman*
30 East 60th Street
New York 22, N. Y.

NINTH ANNUAL MEETING OF THE AMERICAN ACADEMY OF DENTAL MEDICINE

Plans are well under way for the Ninth Annual Meeting of the American Academy of Dental Medicine to be held in New York City at the Park Sheraton Hotel on May 13th, 14th and 15th, 1955. A prominent list of speakers for this convention are now being selected from the ranks of Medicine and Dentistry. The complete scientific and social program for the meeting will be published in the very near future. Plan now to attend the Ninth Annual Meeting in May.

GEORGE J. WITKIN, *Chairman*

PRESIDENTS MESSAGE

GEORGE F. CLARKE, D.M.D.

MY FELLOW MEMBERS—

One of the prime objectives of the American Academy of Dental Medicine, according to our Constitution is "To promote a better scientific understanding between the fields of Dentistry and Medicine."

Because this is a field in which the Academy is particularly equipped to work, I emphasized this subject in the President's Address at the Annual Meeting in Philadelphia. At that meeting, the Dento-Medical Relations Committee, under the able leadership of Dr. Maxwell Goldenberg, submitted an outstanding report. Because I believe that this Report deserves the serious study of every individual member, I have asked to have it reprinted below. Your ideas on implementing this program are requested. Please send your suggestions to Dr. Goldenberg or to me.

REPORT OF DENTO-MEDICAL RELATIONS COMMITTEE

Although a great deal of work has been done in different centers, in Universities and public health departments towards integrating the thoughts of the physician and dentist, there still lies a great unexplored terra incognita between the practices of medicine and dentistry.

A great deal of credit is due to such men as Doctors J. Lewis Blass, Howard R. Craig, John A. Kolmer, Albert L. Midgley, and others for their creating a better understanding between dentistry and medicine. Dr. Craig, speaking in the University of Pennsylvania, had this to say—"Of all the apertures of the body, the mouth is probably the most subject to trauma, infection and general insult. Possibly it is also guilty of committing the most sins. Further, it is more than any of the others, the site of lesions or signs of disease elsewhere in the body. It is rather surprising that physicians in their search for aperture specialization, have thought of the mouth merely as a throughway to the tonsils and points south than as a full station stop with major disease potentialities.

"The rise of oral medicine as a distinct field of interest and specialization is a recent phenomenon for which the public is indebted almost entirely to the dental profession. For the medical profession on the whole has given it relatively little specific thought. It has been one of those things merely included. . . .

"It should be only a natural and much desired phenomenon that the two professions, medicine and dentistry both health services, with common aims, common techniques and interested in a common organism should approximate their fields of work in such a way that the medically trained men should think more in terms in keeping with the importance of the oral cavity as a seat of disease and the dentally trained individual should envisage the whole body while he focuses his immediate attention on diseases of the mouth. . . ."

Dr. Kolmer, who is the director of the Institute of Public Health and Preventative Medicine of Temple University has written numerous articles on this subject.

The Jefferson Medical College and Temple University Medical School are amongst the most important centers at this date, where the operation of a Medical-Dental Clinic for the purpose of correlating and integrating clinical dentistry with clinical medicine are functioning.

This committee, after gathering as much information as is available to them, wishes to recommend the following:

1. To encourage Dental Schools to broaden their courses of instruction in Medicine; to encourage Medical schools to give more information on Dentistry to their medical students.

2. To encourage Medical Associations to have dentists present papers at least at their annual meetings, and have Dental Associations have medical men present papers to them.

3. To encourage Medical and Dental Societies to have mixed groups participate in such programs as questions and answers and symposia.

4. That Dental and Medical schools should have joint lectures on subjects of common interest for their students.

5. To organize Dental-Medical study groups.

6. To organize departments in hospitals for the purpose of correlating and integrating clinical dentistry with clinical medicine.

7. To enlist the support of a governing body in Dentistry and Medicine to plan Dental-Medical Relations programs. (American College of Dentistry, American Dental Association, have appointed such committees to work along these lines).

8. It is the opinion of your committee that our Academy should very actively undertake a program of Dental-Medical Relations. A good start can be made by instructing our local sections to seek the help and cooperate with the local Deans of both medical and dental faculties, hospitals, and local medical societies. There is no doubt that in the higher echelons this problem is recognized and a great deal of thought given to it, but we feel that the average Dental and Medical men have done nothing actively toward common thought and action.

MAXWELL GOLDENBERG, Montreal
Chairman

There are also projects by the Pharmaceutical and the Research Committees that will need your help.

The Pharmaceutical Committee is working on a project for the evaluation of certain drugs used in dentistry. If you are interested in aiding in this study, please contact

DR. THEODORE LITE
1426 Kings Highway
Brooklyn 29, New York

The Research Committee is to present a statistical project on the incidence of periodontal disease. To be of real value, this needs the participation of every member. Please co-operate.

Announcements

POSTGRADUATE TRAINING IN PERIODONTIA AND ORAL MEDICINE NEW YORK UNIVERSITY COLLEGE OF DENTISTRY

The Periodontia Department of New York University College of Dentistry which has been giving postgraduate courses in this subject since 1926, announces its full time course in Periodontia and Oral Medicine for one academic year, September 20, 1954 to May 26, 1955, leading to a certificate. Two year full time, four year half time courses and two year half-time courses are also given.

Ten session (one evening weekly) refresher courses begin Oct. 6th, 1954 and Feb. 23, 1955.

A two week full time course in the Spanish language and a half-year course in the Spanish language are also available beginning January 10th, 1955.

All courses are under the direct supervision of Dr. Samuel Charles Miller.

For information concerning the above courses and other phases of dentistry write to Secretary, Postgraduate Division, New York University College of Dentistry, 209 East 23rd Street, New York 10, N. Y.

GREATER NEW YORK DENTAL MEETING

A "Frontier of Modern Dental Research" will highlight the educational exhibits of the Greater New York Dental Meeting to be held at Hotel Statler, New York City, December 6 to 10, according to recent announcement by Dr. Matthew Besdine, General Chairman.

The "frontier" will present materials, methods and equipment on which researches are now being conducted, including the new Cavitron, an ultrasonic tool for cutting of tooth tissue. Other exhibits in the group will feature electron microscopy of dental tissues, the use of air as a power source for dental handpieces, intra-oral photography, a rapid technic for X-ray development, electronic research, maxillo-facial restorations, and cancer research.

From its opening session, Monday morning, December 6, through to its close on Friday, December 10, at 5:00 P.M., the meeting will present outstanding dental educators in a program designed to provide the utmost in postgraduate education for the general practitioner, Dr. Besdine reported.

Reservations for Hotel Accommodations may be secured through the New York Convention and Visitors Bureau, 500 Park Avenue, New York 22, N. Y. Meeting Headquarters are located in Room 106A, Hotel Statler, New York 1, N. Y.

THIRD PUBLIC HEALTH WORKSHOP

"Health Education and Preventive Dentistry in Industry," is the theme of the Third Public Health Workshop, sponsored by the First District Dental Society of the State of New York with the collaboration of the Postgraduate Medical School, Institute of Industrial Medicine, New York University. The Conference will be held on Friday, January 21, 1955, at the Hotel Statler, New York City.

Dr. Anthony J. Lanza, Emeritus Professor of Industrial Medicine, Post-graduate Medical School, New York University, will address the morning panel on "Health Education in Industrial Dentistry and Dental Public Health." Dean Raymond J. Nagle, of the New York University College of Dentistry, will preside.

Dr. William A. Fennelly, President of the Dental Society of the State of New York, will address the afternoon panel on "Dental Health Education in Private and Institutional Practice." Dr. Edward R. Aston, Industrial Dental Consultant, Bureau of Industrial Hygiene, Pennsylvania Department of Health; Secretary, American Association of Industrial Dentists, will preside.

Following the two panels, summaries and the findings of the Workshop will be presented at a "Discussion-Forum," under the leadership of Dr. Michael S. Burnham, President-Elect of the First District Dental Society of the State of New York.

POSTGRADUATE COURSES OFFERED AT NEW YORK UNIVERSITY COLLEGE OF DENTISTRY BEGINNING OCT. 1954

123A	Periodontia	10/ 6/54 to 12/ 8/54	Friday, 7-9 & 4-6
110A	Complete Dentures	10/ 1/54 to 12/10/54	Friday, 1-7
121	Pathology	10/ 8/54 to 12/17/54	Friday, 1-4
101	Anatomy	10/13/54 to 12/ 1/54	Wednesday, 7:30-10
129A	Roentgenology	10/14/54 to 11/11/54	Thursday, 4-6
113A	Operative Dentistry	10/15/54 to 2/11/55	Friday, 4-7
107	Crown & Bridge	10/15/54 to 1/21/55	Friday, 2:30-7:30

For information write: Secretary, Postgraduate Division, New York University College of Dentistry, 209 East 23rd Street, New York 10, N. Y.

NEW YORK UNIVERSITY PERIODONTIA ALUMNI ASSOCIATION

The Periodontia Alumni Association of New York University has just been formed. Membership is open to all dentists who have taken postgraduate courses in Periodontia at the university. Some of the aims and objectives of the Association are: to have regular meetings where views can be exchanged and where cases and techniques can be discussed; to provide opportunities to present papers and clinics, and to stimulate and encourage research in Periodontia.

For further information and a membership application, please write to Dr. J. Robert Theaman, Chairman, 2 East 54th Street, New York 22, N. Y.

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